JOURNAL
OF THE
AMERICAN VETERINARY MEDICAL ASSOCIATION

(Contents continued on adv. pages 2 and 4)

Volume 127 OCTOBER 1955 Number 943

vaccinate feeder lambs against Type D enterotoxemia

Clostridium Perfringens Type D Bacterin *

Most sheepmen report that vaccination against Type D (ovine) enterotoxemia is essential for successful feeding operations. Otherwise, heavy losses may occur.

Millions of lambs have been protected by practitioners who immunized them with Corn States Clostridium Perfringens Type D Bacterin.

Vaccination with Type D bacterin prevents enterotoxemia of sheep either on pasture or in the feedlot. It prevents losses, saves feeding time, and gets the lambs to market early.

This season vaccinate feedlot lambs with Type D bacterin. After the expiration of the 10 day period for development of immunity, sheepmen can allow access to lush pastures or fattening rations with little danger. Type D bacterin is available in 50 cc., 250 cc., and 500 cc. bottles.

Clostridium Perfringens Type D Antitoxin

Check explosive outbreaks of enterotoxemia with Clostridium Perfringens Type D Antitoxin. Type D antitoxin establishes immediate passive immunity. It can be given to range, feeder, and suckling lambs. Type D antitoxin is available in 100 cc. and 250 cc. bottles.

Type D bacterin and Type D antitoxin were originated and first introduced to practitioners after years of research in Corn States laboratories. They have received enthusiastic and unqualified endorsement from the profession and from the sheep industry. They are sold to graduate veterinarians only. Write for literature.

*Patent 2,719,102

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CONTENTS

(Continued from Cover)

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SURGERY AND OBSTETRICS

$10.00 per annum **Foreign $10.50; Canada $10.50** **Single Copies $1.00 Prepaid in U.S.**

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for use in

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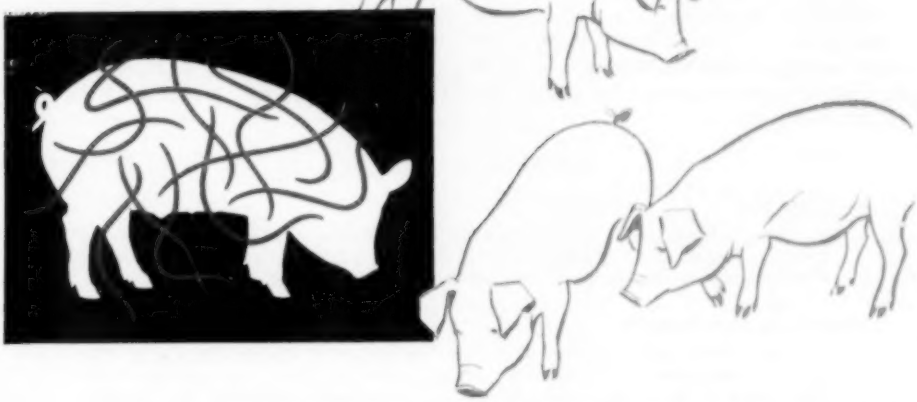

Aska-Rid has the unique advantage of being BOTH safe and effective for eliminating roundworms from debilitated swine.

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CONTENTS—Continued

CLINICAL DATA

CURRENT LITERATURE

THE NEWS

MISCELLANEOUS

M·L·V is recommended for use in a 2 cc. dose for healthy, unexposed swine of any age over 5 weeks, with serum dosage of 10 cc.

M L V is the trade mark of Fort Dodge brand of modified live virus hog cholera vaccine. (The letters MLV should not be applied indiscriminately as an abbreviation of the term "modified live virus.")

•

M L V is prepared from swine tissues, including blood, selected from pigs inoculated with virus modified by serial passage in rabbits. (U.S. Patent No. Re. 23746).

•

M L V is used without serum on susceptible pigs in potency and safety tests and experimental field vaccination. Such use in routine vaccination is not recommended, however, because of occasional complicating factors not easily recognized nor controlled.

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M L V was formulated from its beginning for simultaneous administration with anti-hog cholera serum.

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M L V is recommended for use in 2 cc. dose for healthy, unexposed swine of any age over 5 weeks, with serum dosage of 10 cc.

With M L V, amounts of vaccine and serum are not correlated. Vaccine dosage is standard but serum dosage should be varied to meet requirements of each individual animal or herd. Larger doses should be used when, in the opinion of the veterinarian, they are indicated.

•

M L V, when used as recommended, provides positive protection against hog cholera throughout the lifetime of average swine — even up to 25 months.

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M L V is thoroughly tested to establish safety and freedom from danger of infecting the premises with hog cholera or other swine disease.

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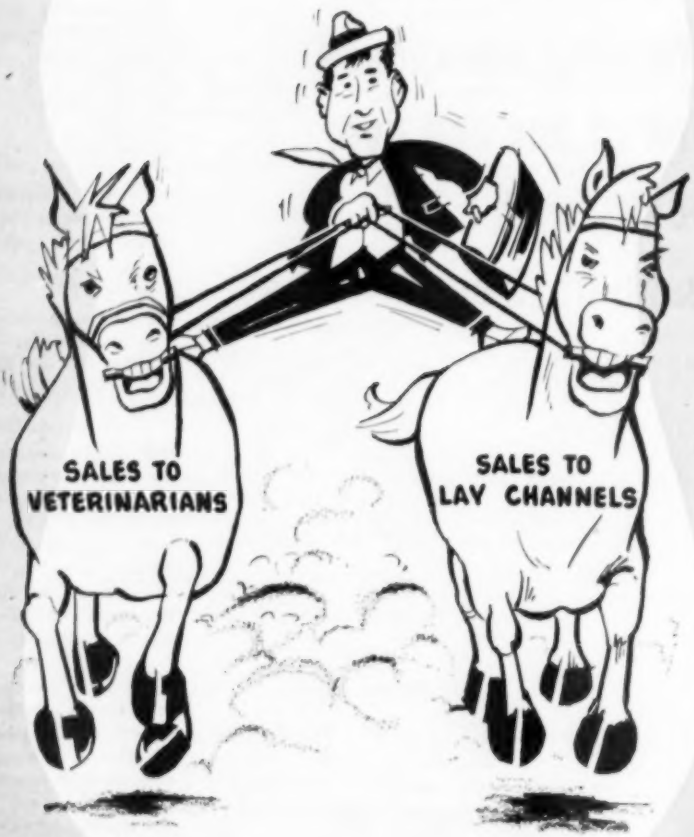

Straddlers who try to ride the "professional" horse with one foot and the "lay" horse with another are in for a bad fall in this business.

The wise veterinarian knows that the supply house which really seeks to serve the veterinarian is on the veterinarian's side 100 %. There is no place for straddling. No compromising. No jumping from one side to the other.

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AVMA ☆ Report

Veterinary Medical Activities

✦ The Ninety-Second Annual Meeting in Minneapolis, Aug. 15-18, 1955, set new records in attendance, excellence of program, number and quality of scientific and commercial exhibits, and in general interest.

* * *

✦ The registration totalled over 3,700, about 200 more than the Toronto meeting in 1953 which topped the record up to that time. A detailed breakdown of the attendance at the Minneapolis meeting will be published in the November JOURNAL.

* * *

✦ The AVMA was host to student chapter and student auxiliary delegates to the AVMA Convention in Minneapolis at a dinner held in the Radisson Hotel on Sunday evening, August 14. Officers of the AVMA and AVMA auxiliary spoke to the delegates following the dinner.

* * *

✦ Lt. Colonel Norbert A. Lasher, V.C., U.S.A.F., represented the AVMA at the Annual Congress of the British Veterinary Association held in Belfast, Northern Ireland, September 4-10. Colonel Lasher is presently stationed in England.

* * *

✦ President Floyd Cross appeared on the program of the New Mexico V.M.A. held at Albuquerque on September 12-13.

* * *

✦ On September 27-28, Dr. Hardenbergh and Dr. Kingman met with the Committee on Local Arrangements for the 1956 meeting of the AVMA to be held in San Antonio, Texas.

* * *

✦ President Floyd Cross visited the Maxwell and Gunter Air Force Bases in Alabama, July 28-29. Dr. Cross is presently serving as National Civilian Veterinary Consultant to the Surgeon General of the U.S.A.F.

* * *

✦ Dr. George H. Hart and Dr. Peter Diplock, AVMA members in Australia, represented the AVMA at the Australian V.M.A. meeting in Sydney, May 30 to June 2, 1955.

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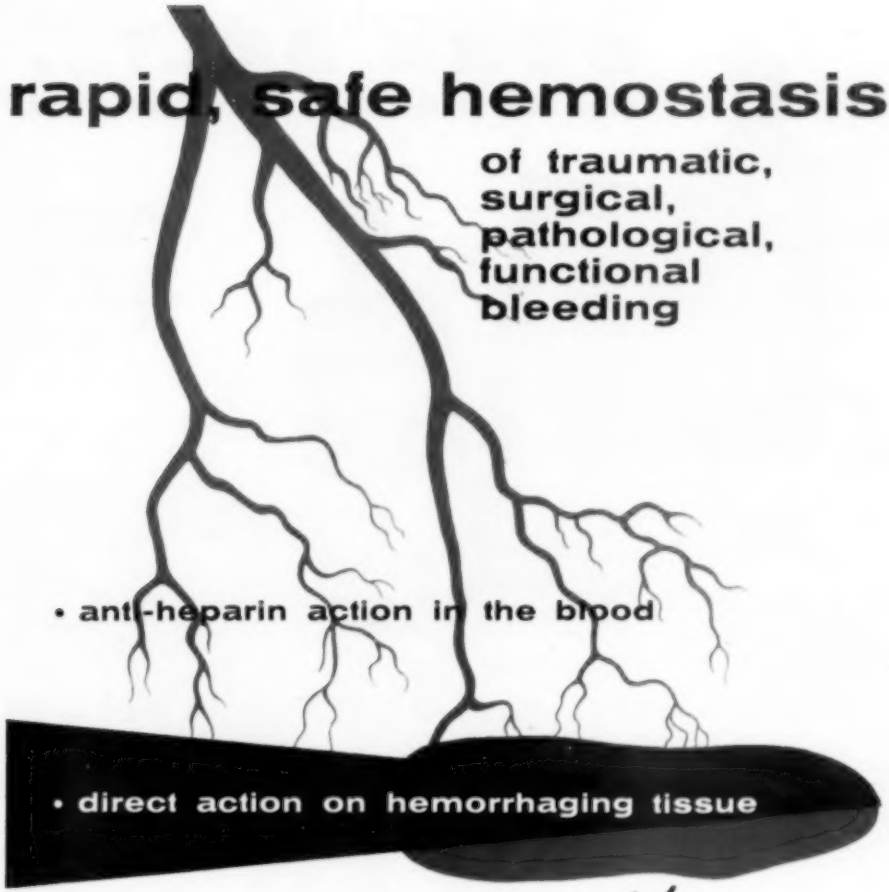

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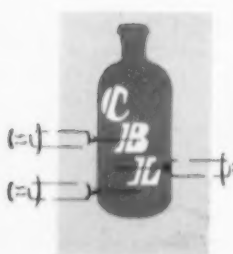

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1. Choy, D. S. J., and Wendt, W. E.: A New Local Treatment of Burns. U. S. Armed Forces M. J. 3:1241 (Sept.) 1952.
2. Watts, R. E.: A New Plastic Surgical Bandage. J. A. V. M. A. 125:40 (July) 1954.
3. Wendt, W. E.: Personal communication. (Sept.) 1954.

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References: 1. Brown, A. L., et al.: To be published. 2. Agricultural Research Service, Losses in Agriculture, June 1954, Table 20, p. 129.

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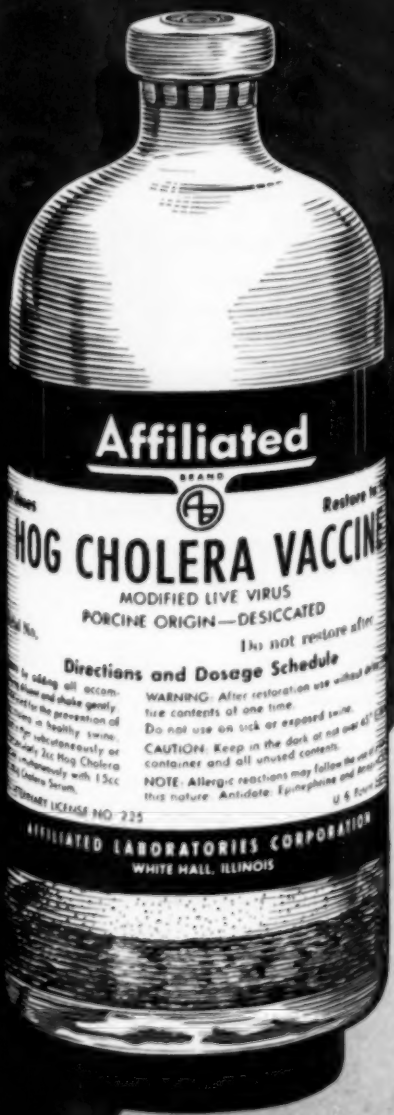

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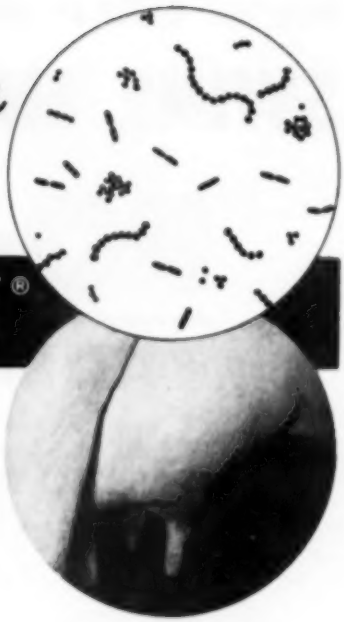

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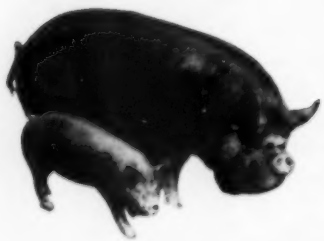

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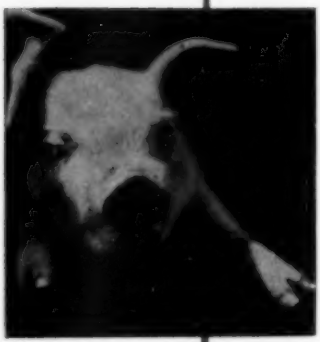
2. **As a primary treatment for swine bacterial enteritis in herds showing no systemic reaction.**
3. **As a complementary treatment for swine bacterial enteritis in herds showing systemic reactions.**
4. **To minimize relapses.**
5. **To minimize stunting effects following the outbreak of systemic diseases.**

ARMOUR



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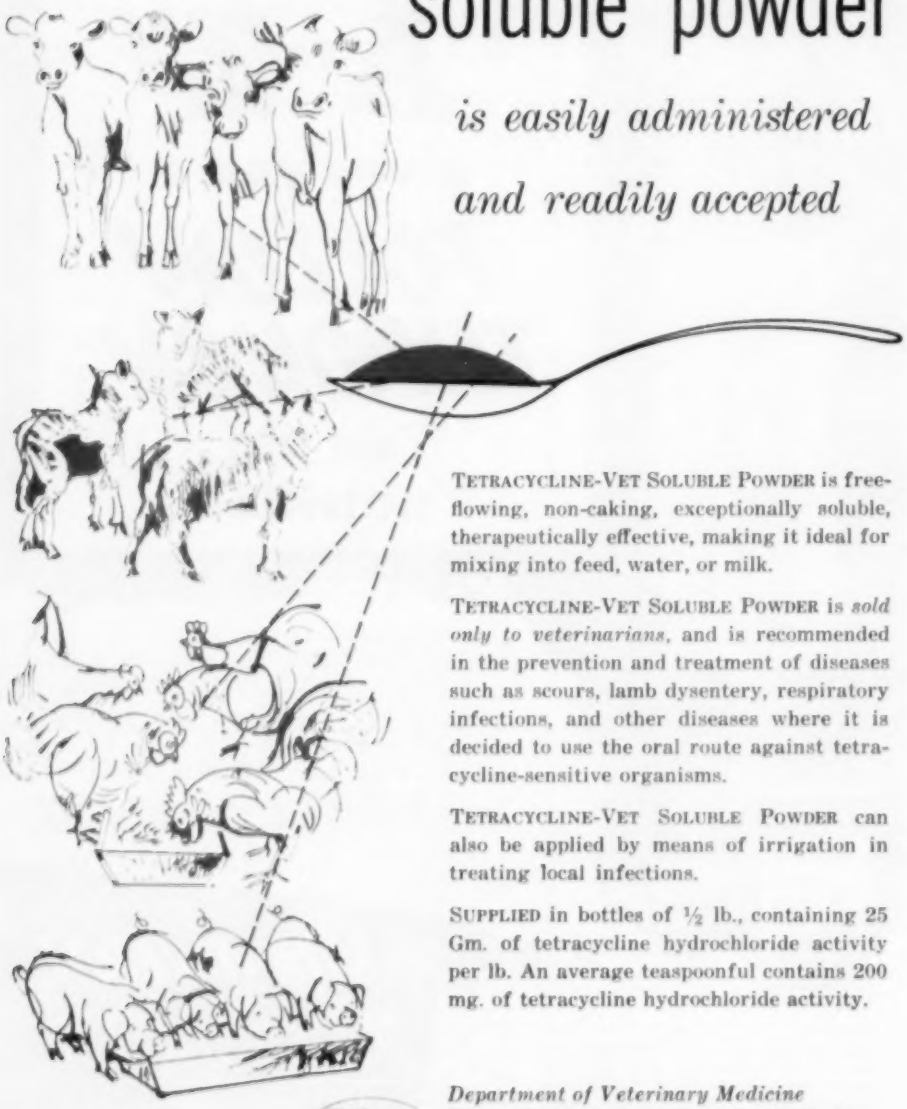

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Bottle of 100.

Fort Dodge Laboratories, Inc., Fort Dodge, Iowa.

Journal
of the
American Veterinary Medical Association

VOL. 127 OCTOBER, 1955 No. 943

The President's Address

A. H. QUIN, D.V.M.

Kansas City, Missouri

This Ninety-Second Annual Meeting of the AVMA promises to shatter previous attendance records and this is as it should be, for it reflects the rapid advancement, development, and technological progress of our profession in America.

We have every right to be justly proud of this almost century-old professional association, for its membership is now nearing the 14,000 mark, and 97.3 per cent of its members renew their memberships annually.

I am sure that you will be pleased to know that our executive office reports an enrollment of 1,037 new members during the past fiscal year.

We can be equally proud of our fast-growing Women's Auxiliary with its 4,000 or more members.

Now, through these many years, I have heard diverse discussions on the public relations aspects of veterinary medicine. My dominant conclusion is that, next to the quality of services rendered by veterinarians, the most influential force in attaining and extending a favorable public acceptance and appreciation of our profession is the position of local community leadership held by the wives of veterinarians in hundreds of towns and cities throughout the United States and Canada. And we should be deeply grateful to the Women's Auxiliary of the AVMA for its continued enviable record in intraprofessional activities.

It is historical custom that your president shall outline, at this plenary session, matters that directly or indirectly appertain to the welfare of veterinary medicine in America, make specific recommendations, and cite highlights of the Association's current activities.

However, I consider it my first duty, before this audience, to pay particular tribute to AVMA members who have worked so diligently and capably on our many committees, councils, and special assignments this past year—efforts that have contributed materially to the continued progress of our Association. These men have unselfishly taken time from busy careers and practices to serve our needs and I am sure you agree that we owe them a heartfelt vote of thanks.

Lack of time precludes even a highlight

Dr. A. H. Quin

review of their many accomplishments. Their reports will appear in the forthcoming "Proceedings Book" and should be read and studied by every member. The chair specifically requests that you review the notable progress reports of the Committee on Brucellosis, the Committee on Foreign Veterinary Graduates, the Committee on Motion Pictures and Audio-Visual Aids, the Committee on Veterinary Services, and several others which I will mention later in this address.

We are also grateful to Past-President General McCallam for his unstinting efforts and accomplishments as Washington representative of the AVMA.

If time permitted I could, by a simple report of facts, greatly fortify your pride and appreciation of the efficient handling of increasingly complex duties by the outstanding executive officers and personnel of our office in Chicago.

And, on behalf of the entire Association, we sincerely appreciate and thank all members of the Local Committee on Arrangements and their wives for the thousands of aggregate hours of hard work they have contributed in planning this major meeting. As Walter Winchell says, "An orchid to every one of them."

THE FUTURE NEED FOR VETERINARIANS

As previous presidents have emphasized, we must face our full responsibility to supply an ample complement of technically trained veterinarians in all branches of veterinary service to the two great nations of America. The greatest tragedy that could possibly befall our profession is that we might fail in meeting this responsibility.

Let us realize that there are still approximately 1,000 counties in the United States and Canada that are entirely devoid of services by graduate veterinarians. There are, in addition, many specific areas where qualified veterinary service is too sparse to be economically practical.

Our most skilled economists predict a necessary 35 per cent minimum increase of animal protein foods to feed a continental population of 225,000,000 within the next 20 years. In addition, and as good neighbors, we will be called upon to supply an ever-increasing amount of foods of animal and poultry origin to many of the less-productive nations of the world. To abridge all technical and statistical explanations, this simply means that greater productivity and efficiency in breeding, husbandry, feeding, disease prevention, and

Retiring President A. H. Quin (left) receiving the service scroll for a year of work well done, from Chairman of the Board S. F. Scheidy.

treatment are mandatory if we are to meet production goals of the future.

Likewise, the unprecedented migration of America's population from urban row houses, tenements, and apartments to the "shopping center" type of suburban life, directly relates to the future need for veterinarians. Families who move to a home and a yard of their own produce two primary crops, namely, babies and pets. Market surveys indicate an increase of our dog population from 22,000,000 to 30,000,000 or more within ten years. This means an ever-increasing need of veterinary graduates for small animal practice—a factor which must be carefully considered in evaluating the projected number of general practitioners needed.

It is sometimes forgotten that, in addition to supplying an adequate complement of all types of practitioners, our schools must also fill the need for veterinarians in the armed forces; research; federal, state, and municipal food inspection services; livestock regulatory services; for biological and pharmaceutical production; "Point Four" foreign service; educators in veterinary colleges and extension services; zoos, fur farms, and a rapidly growing list of positions in the public health services and industry.

To meet all of these requirements, we are graduating a scant 1,000 veterinarians a year from our 19 continental colleges—less than 600 new replacements over deaths and retirements annually.

Your president makes the specific recommendation that the executive officers and Executive Board of the AVMA take definitive action to adequately and properly evaluate the projected need for veterinary personnel over the next 25 years.

Official Services

Currently, the relations between practitioners of the states and the Agricultural Research Service of the U. S. Department of Agriculture are greatly improved and distinctly more progressive than in the past. The committed policy of the federal services under the leadership of Drs. Van Houweling, Clarkson, Simms, and others is to place a maximum amount of cooperative federal-state animal disease control in the hands of private practitioners. A current example is the widespread activation of

Incoming President Floyd Cross (left) receives gold key from Chairman of the Board S. F. Scheidy.

private practitioners in the accelerated brucellosis control program—a campaign which has already made signal progress in eradicating this plague of man and livestock.

Some of our states (e.g., California, Ohio, New York, largely through the efforts of planned veterinary leadership, have developed livestock sanitary services of enviably high standards. Hence, it is doubly regrettable that other states continue to drag along with a "horse-and-buggy" type of livestock disease regulatory service. In some of these states, the state veterinarian is appointed on a purely political patronage basis, with no regard whatever to his technical or administrative qualifications. Furthermore, the salary scale is sometimes less than that paid semi-skilled labor in industry.

It is a fixed professional obligation that our constituent state associations join other organized state livestock organizations and clean up this archaic, inadequate, and incompetent situation as soon as possible.

PRACTICE ACTS

A kindred problem is that the veterinary practice acts of some states have not been revised for as long as 50 or 60 years. Even some of the less ancient practice acts are inadequate in their true purpose, *i.e.*, to protect the public from mercenary and unqualified charlatans. Neither do most of these state practice acts, or allied state regulatory measures, protect farmers from the abuses of commercially vended live viruses, bacteria, and potentially dangerous drugs.

Immediate revision of state practice acts is an urgent need that calls for maximum coordinated effort between the AVMA and state associations. Your president recommends that a special committee be appointed to: (1) prepare a model state practice act or acts that can be supplied to constituent associations on request; and (2) prepare an outline of procedure for guidance of state associations in legislative activity aimed at practice act revisions.

REGIONAL RESEARCH LABORATORIES

We know that this nation's livestock interests contribute over $20 billion a year to the national income. We also know that disease and parasite losses exceed $2 billion annually. We see countless millions of dollars allocated to foreign aid of all types. We also see the ridiculous allocation of less than $1.00 for each $1,000 of net husbandry revenue allocated to animal disease research. We see first-hand the futility of indiscriminate allocation of federal funds to "catch as catch can" disease research at experiment stations and the reluctance of many state legislatures to supply little other than token funds for animal and poultry disease research.

An immediate need is for the U. S. Department of Agriculture, by congressional authorization under the Bankhead Act, to establish adequately staffed and separate regional laboratories for swine, cattle, and sheep disease research. Your president urges activation of legislative pressure and action at all levels to attain this objective.

COMMERCIAL QUACKERY

The facts are irrefutable that antibiotics and selective sulfonamides are invaluable in saving both human and animal lives. The facts are also irrefutable that nationwide overexploitation of these agents to untrained laymen has posed many vexatious problems both to the veterinary profession and to the livestock industry. The same can be said for intensive exploitation of "shelf goods" markets for viable hog cholera vaccines, anthrax spores, disease-transmissive poultry vaccines, and other potentially dangerous products. The corporate interests which promote the precept that every farmer can be his own animal doctor also spend money lavishly in propagandizing our veterinary colleges, livestock regulatory personnel, and rural educational agencies. They expend intensive sales effort on our practitioners to convert professional dispensing into open display merchandising and questionable promotion methods.

Correction of this complex and multi-angled problem is essential, not only as a protective measure for veterinary practice, but for the long range good and protection of the nation's livestock industry.

Your Association's Special Committee on Veterinary Supply Problems, under the chairmanship of Dr. Frank Young of Iowa, is charged with the responsibility of investigating all angles of infringement on veterinary practice by external interests. Your president recommends that all con-

stituent and provincial associations appoint special committees to coordinate activities with Dr. Young's committee at the continental level. It is further recommended that the Executive Board provide a special item in the budget, if necessary, to insure maximum activation of the Committee's (Veterinary Supply Problems) efforts at both national and state levels.

PUBLIC HEALTH

Science is rapidly unfolding additional evidence of viral, bacterial, and parasitic diseases that are common to man and animals or actually transmissible from animals to man.

The broad scope of technical subjects to be presented at the Public Health Section of this meeting is specifically indicative of the intricate relationship of veterinary science to the public health. We are also cognizant of the role of veterinary scientists in establishing criteria for protection of man from the effects of irradiation and atomic fallout by study on experimental animals. Some 40 veterinarians are already assigned to this ultratechnical field of atomic research.

The inclusion of one or more veterinarians on the technical staff of state and provincial public health services is an essential step in the right direction.

Your president recommends that the AVMA Committee on Public Health and Zoonoses, together with allied AVMA committees and our executive officers, exert all possible cooperation to constituent associations in placing more full-time veterinarians on state and previncial boards of health.

INSURANCE

Your Special Committee on Insurance has worked hard this past year in surveying the possibilities of obtaining national pure group health and accident insurance for AVMA members. If this goal is attainable, it should effect many advantages and bring preferential health and accident coverage to every AVMA member under 70 years of age in all states and provinces at attractive premium rates. We will anxiously await results of the decision of the Executive Board and House of Representatives on the insurance committee's report at this session.

Your president specifically recommends that the Special Committee on Insurance shall be continued, and that it shall be given the status of a standing committee at the discretion of the Executive Board and House of Representatives.

CONCLUSION

In conclusion, I am humble and grateful to all of you for the opportunity to have served as your president-elect and president. I have attended many veterinary meetings and met with many of the AVMA student chapters. I sincerely thank all veterinarians and their wives for the countless courtesies they have extended to me during attendance at their meetings. My end conclusions are that veterinary medicine is growing and expanding rapidly; that both urban and rural populations are increasingly appreciative of the role played by our profession; and that the future is indeed bright for the ever-expanding utilization of veterinary science and practice services.

Highlights of the Ninety-Second Annual Meeting, Aug. 15-18, 1955 in Minneapolis

Excerpts from Reports of AVMA Staff

Executive Secretary J. G. Hardenbergh reported a net gain in membership of 737 for the past year. The AVMA on Aug. 1, 1955, had 13,847 members of which 93 per cent have paid current dues. Dr. Hardenbergh reported that the AVMA now occupies about 5,600 square feet of office space, has a staff of 22, of whom 20 are full-time employees.

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Assistant Executive Secretary H. E. Kingman, Jr., outlined several of the AVMA programs. Committee, legislative, and convention activities were highlighted and a brief summary of AVMA film library and student chapter progress was reported.

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Brigadier General J. A. McCallam, AVMA Washington representative, reported on the activities of the Washington office. The contacts maintained with government agencies and departments, the work of this office on legislative matters, and the outline of routine operations were given by General McCallam. Mention was made of 14 House and Senate bills of special interest to the veterinary profession.

• • •

Dr. W. A. Aitken, editor-in-chief, reported that AVMA publications contained a total of 2,156 pages of reader material (exclusive of advertising) during the calendar year 1954. Dr.

Dr. B. T. Simms (right), director, Livestock Research, ARS, U.S.D.A., Washington, D. C., accepting the Twelfth International Veterinary Congress Prize from President Quin at the Ninety-Second Annual Meeting of the AVMA in Minneapolis.

Dr. H. E. Kingman, Sr. (right), received the Borden Award at the Minneapolis meeting. Mr. John H. McCain (left), of the Borden Company Foundation, and President A. H. Quin congratulate him.

Aitken also reported that Dr. Wayne H. Riser had been employed as editor (part-time) for small animal medicine.

• • •

Helen S. Bayless, assistant editor and advertising manager, reported on plans to accomplish the smooth production of the JOURNAL on a semi-monthly basis, beginning with the Jan. 1, 1956, issue, including the employment of a fourth editorial assistant. Mrs. Bayless also mentioned the publishing, as Part II of the Research Journal (Oct., 1955), of the bibliography of "Diseases in Wild Mammals and Birds," prepared by Dr. Patricia O'Connor Halloran, and the production of a 24-page "Style Guide" this year.

The circulation of the JOURNAL as of June 30, 1955, was 17,735, and of the *American Journal of Veterinary Research*, 3,523.

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Mr. R. G. Rongren, director of membership services, reported that 98.5 per cent of those receiving a 1955 dues notice last November have renewed their memberships this year. This is slightly more than 1 per cent higher than renewals received last year for the same period.

Mr. Rongren also reported on the activities of the circulation, directory, subscription, application, and order departments, and pointed out that this phase of AVMA activities has grown tremendously during recent years. Streamlining and improving major systems and procedures will be given increased attention during the coming year.

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Treasurer H. E. Kingman, Jr., reported that receipts to the AVMA General Fund during the fiscal year amounted to $399,582; expenses totaled $380,119.97, resulting in a net gain in assets of

Dr. C. W. Bower (right), Topeka, Kan., was the winner of the AVMA award. President Quin presents it to him.

President Quin accepts the Founder Centennial Award of Swift & Company awarded to the AVMA "for outstanding leadership in the livestock and meat industry." The award was presented by Dr. H. E. Robinson, director of laboratories, Swift & Company.

$19,462.03 during the year. The Research Fund operated on a "break-even" basis during the year — receipts $10,472.81, expenses $10,482.23.

Mrs. A. H. Quin receiving flowers presented to her by the Minneapolis Local Committee on Arrangements at the Ice Show on Tuesday evening during convention week. President Quin smilingly looks on.

Mrs. L. R. Richardson, president of the Women's Auxiliary to the AVMA, addressing the Opening Session of the Ninety-Second Annual Meeting.

Dr. I. D. Wilson (left), Blacksburg, Va., accepting the Humane Act Award for Martha Ann Coppedge, 10, Portsmouth, Va., from Dr. G. W. Mather, chairmen of the Humane Act Award Committee, at the Opening Session of the AVMA Annual Meeting in Minneapolis. Inset—Martha Ann and "Buffy," the Cocker Spaniel she rescued from an icy lake last February.

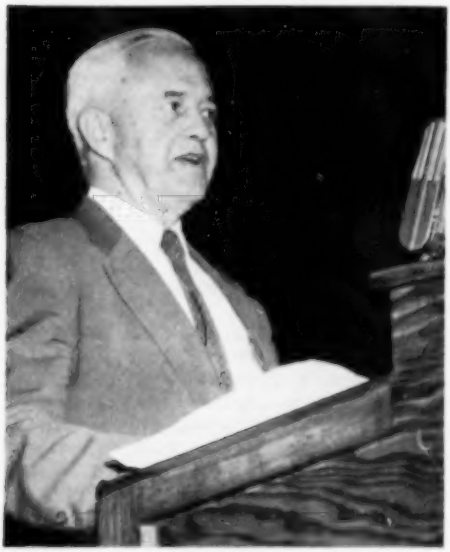
Preconvention Conference on Veterinary Medical Examination and Licensure

Over 200 members of state and provincial examining boards, association secretaries, members of veterinary school faculties, livestock sanitary officials, and others concerned with education and licensure attended the preconvention conference held on Sunday, August 14, preceding the ninety-second annual AVMA meeting in Minneapolis. The program was in charge of Dr. C. W. Bower, Topeka, Kan., and Dr. J. G. Hardenbergh, Chicago, president and secretary, respectively, of the National Board of Veterinary Medical Examiners.

During the morning session, various aspects of professional examination and licensure were discussed by Drs. A. H. Quin, C. W. Bower, W. R.

President-Elect Wayne O. Kester is installed, by President Quin, at the Closing Session.

Treasurer H. E. Kingman, Jr. (left), is installed by President Quin at the Ninety-Second Annual Meeting of the AVMA.



Incoming President Floyd Cross addressing the Closing Session after his installation by Retiring President A. H. Quin at the Minneapolis meeting.

Krill, Lillian D. Long, H. E. Kingman, Jr., and Mr. Sam Mayo. (*See* program, page 15, July JOURNAL.)

At the luncheon, Dr. Dean F. Smiley, secretary, American Association of Medical Colleges, reviewed briefly the history of medical licensure and pointed out some of the current problems and needs in measuring professional competence in the healing arts.

The afternoon session heard presentations from Drs. L. J. Goss, L. C. Payne, A. Zeissig, G. R. Cooke, and J. V. Smith. Dr. W. T. S. Thorp then conducted a general discussion period which emphasized and summarized the program contributions.

This is the first time in many years that the various fields of veterinary medical examination and licensure have been discussed comprehensively at an AVMA convention; the interest in this conference was such that a number of requests for similar programs at future meetings were received.

The complete proceedings of the Minneapolis preconvention conference will be published in "Proceedings of the Business Sessions" of the 1955 annual meeting as Part II of either the December or January JOURNAL.

General Kester Is President-Elect

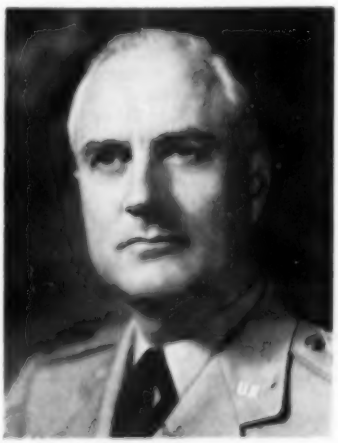
Brigadier General Wayne O. Kester, chief of the Air Force Veterinary Service, was unanimously elected to the office of president-elect of the AVMA at the opening session of the Ninety-Second Annual Meeting in Minneapolis, August 15. He succeeds Dr. Floyd Cross who was installed as president at the final session of the convention on August 18.

General Kester was born Aug. 27, 1906, at Cambridge, Neb., where he received his schooling, graduating from high school in 1925. After attending Nebraska State Teachers College at Chadron, Neb., for two years, he enrolled at the School of Veterinary Medicine, Kansas State College, graduating with honors in 1931.

For two years prior to graduation, he worked in the poultry disease laboratory at Kansas State. After graduation, he participated in general practice for two years, working with range cattle in Kansas for a time, then chiefly with small animals in Akron, Ohio.

He was commissioned as second lieutenant in the Regular Army Veterinary Corps in July, 1933. He served first as district veterinarian with the Civilian Conservation Corps at Fort Hayes, Ohio, then at Elkins, W. Va. After six months he entered the Army Medical Field Service School at Carlisle Barracks, graduating in June, 1934. He then served for three years with the First Cavalry Division at Fort Bliss, Texas, until assigned to the Army Medical Center at Walter Reed Hospital, Washington, D. C., in June, 1937. As a captain, he served as port and depot veterinarian at Seattle, Wash., during 1938 and 1939, and at Hawaii during 1940 and 1941. He witnessed the start of the attack on Pearl Harbor, Dec. 7, 1941, from a hillside while on his usual early Sunday morning trail ride.

During all of World War II, General Kester



President-Elect Wayne O. Kester

served as chief veterinarian, U. S. Forces Pacific Ocean Areas (unified services), supervising the establishment and maintenance of veterinary service on numerous islands. He was advanced to lieutenant colonel in December, 1942, and to colonel in July, 1943. He simultaneously served as staff advisor to the Military Governor and later to the Governor of Hawaii, for which he received special commendation from the Governor. In August, 1945, he was assigned as chief of the Meat and Dairy Hygiene Branch, Office of the Surgeon General, Washington, D. C., serving under General McCallam.

In July, 1949, he was transferred to the Air Force as director of the newly established Air Force Veterinary Service. This service started with about 60 veterinarians and now includes some 300, about a third of whom are overseas. The duties of the service include: assuring the quality and wholesomeness of all foods for our global Air Force as well as assisting in the over-all public health and research programs. Integration with the medical and other services is stressed. General Kester, who became a brigadier general on July 18, 1953, received special commendation from the Surgeon General, U. S. Air Force, for developing this unique service.

General Kester was raised in the cattle country in western Nebraska, where he has long been an owner of a commercial beef herd. His interest in the swine industry started when, at the age of 16, he purchased a 4-H gilt and won a "ton litter" contest. This venture developed into a partnership with his father and, a few years later, into a 500-sow herd producing about 5,000 pigs per year.

When in high school, General Kester played football, and at Nebraska State Teachers College he was an all-conference guard on the 1926 conference championship team. He is an enthusiastic horseman and is well known as a horseshow judge in both eastern and western horse circles. He enjoys trail riding and was winner of several equestrian contests during an eight-day ride with the Rancheros Vistadores, California (700 riders), in May of 1955.

General Kester is the author of numerous articles on military veterinary medicine and is a regular contributor to *Horse Lovers Magazine*. He was awarded the Legion of Merit and Army Commendation Medal for his wartime service. He has been a member of the AVMA since 1931 and is affiliated with many other groups, including the American Public Health Association, Association of Military Surgeons, and the U. S. Livestock Sanitary Association.

He was married to Inez Hill, a Kansas State College graduate in home economics, in 1933. His brother, Howard (KSC '35) is practicing veterinary medicine in Cambridge, Neb., and his nephew is a veterinary student at Kansas State College.

His responsibilities as chief of the Veterinary Service of the Air Corps have given General Kester a unique perspective of veterinary medicine, since the world-wide animal disease and food supply situations must be kept under surveillance. This is necessary, both for the possible effect on the health of the troops overseas and to prevent the introduction of foreign diseases into the United States.

Vice-Presidents Elected at Minneapolis

The vice-presidents for the coming year, who are selected on the basis of one from each of the AVMA convention areas, were unanimously elected as follows:

Zone 1, Dr. J. H. Krichel (CVC '16), General Practitioner, Keokuk, Iowa.

Zone 2, Dr. Oscar Sussman (MSC '40), Public Health Service, Princeton, N.J.

Zone 3, Dr. M. R. Blackstock (COL '10), General Practitioner, Spartanburg, S. Car.

Zone 4, Dr. John D. Stevens (WSC '30), General Practitioner, Sequim, Wash.

At large, Dr. A. Grant Misener (ONT '38), Small Animal Practitioner, Chicago, Ill.

Myxomatosis in Britain

The 1954 report of the Advisory Committee on Myxomatosis indicates that, in many areas, 99 per cent of the wild rabbits had been destroyed. The benefits, such as a decrease in lost crops and success in planting trees without fencing, have stimulated organized cooperation between government officials, farmers, and others for the elimination of the few survivors. Objections have come only from a few sportsmen and a few with a "humanitarian angle."—*Vet. Rec., July 9, 1955.*

Production of Feeder Pigs

So-called "pig hatcheries" have had their problems, including disease prevention. The *Prairie Farmer* (May 21, 1955) reports that many of these problems have been overcome by a feeder pig-marketing association which has the pigs raised in northern Wisconsin, where few hogs have been raised in the past, by dairy farmers who are glad to have a use for their skim milk and whey. As orders are received the pigs are delivered at a set place and price, and the farmer is paid cash. These pigs which have been vaccinated for cholera and treated for worms are inspected by veterinarians and given a 10-cc. dose of hog cholera serum, after which they are delivered directly to the purchasers, most of whom are in the adjoining states.

Magnets in the Control of Traumatic Gastritis

A Preliminary Report

R. E. CARROLL, D.V.M.

Buena Park, California

Bovine traumatic gastritis, in some areas, is one of the most, if not the most, important problem in dairy practice today. It is easily confused with numerous other conditions affecting cattle. Rumenotomy, the treatment of choice, appears to be economically sound when the cases are properly selected on the basis of history and hematology.

However, since prevention is better than treatment, we became interested in Cooper's[1] proposed use of alnico bar magnets as a control procedure. As resident veterinarian for a large dairy concern, I had performed over 200 rumenotomies in a year, which indicates the severity of the problem. Therefore, Cooper-type magnets, 2½- by 1-inch cylinders, were purchased.*

The investigation was conducted on first-calf heifers as they entered the milking herd. Since they are maintained on pasture from 4 to 6 months of age until heavy with calf, the amount of "hardware" in their stomachs should be at a minimum at that time. Also, with such an age group the investigation could be continued for a greater length of time than with older animals.

The magnets were administered *per os* with a balling gun after the heifers received herd numbers upon freshening. In a group of 100 heifers, 42 were selected at random and each of these received a magnet on December 16 or 30, 1954. The remaining 58 heifers served as controls. All of the heifers were then placed in heifer strings and were handled and fed in like manner and were equally exposed to ingestion of foreign material.

Results and Discussion

In the first six months of investigation, or until July 1, 1955, 33 of the 58 control heifers (57%) required rumenotomies. Foreign body items such as haywire and nails in the process of penetration were recovered in all instances. This rate was unusually high, the reason in part being the improper functioning of magnets in the grain mixer during at least two months of this same period. However, only 2 of the 42 heifers with magnets (5%) have required operations. Surgery was performed on 1 heifer one month after she received the magnet and the offending wire must have been in her reticulum for two months as it was characteristic of pieces found in chopped green alfalfa, none of which had been fed in that period. In this heifer, the magnet was found attached to the penetrating body. In the other heifer, the offending object was a nail which was penetrating the rumen wall while the magnet lay on the floor of the reticulum.

At the end of the six-month period, it was decided to perform a rumenotomy upon one of the test heifers to determine the quantity of "hardware" picked up by the magnet. The heifer selected had remained

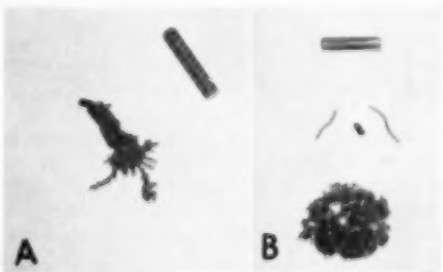


Fig. 1—(A) Unused magnet (above) shown with the magnet recovered from test heifer. The attached foreign material is shown partly spread out for display purposes. (B) Magnet (above) taken from test heifer, separated from its yield of 2 wires, a piece of ferrous material, and iron filings.

Dr. Carroll was resident veterinarian at the Roger Jessup Farms, Glendale, Calif.

*Alnico V-part 27454, the Indiana Steel Products Co., Valparaiso, Ind.

[1]Cooper, H. K.: A Proposed Procedure for Controlling Traumatic gastritis. J.A.V.M.A., 125, (1954): 301.

normal and had terminated her first lactation. The magnet was recovered from the floor of the reticulum with two wires, a piece of ferrous material, and numerous iron filings attached (fig. 1).

The wires were lying flush with the magnet and with no overlap. No foreign objects other than those attached to the magnet were found and no reticular adhesions were present. A mechanic's hand magnet, which is used routinely in rumenotomies, was passed over the interior walls of both reticulum and rumen with negative findings. Apparently, the magnet had attracted all of the "hardware" ingested by the animal during this lactation.

CONCLUSION

The alnico magnet is apparently effective in the prevention of traumatic gastritis of cattle. While a six-month period is not sufficient to warrant any positive statements, the results were encouraging. Approximately 300 cattle in the herd mentioned are now carrying magnets so a more complete report will be possible in the future. We feel definitely that this traumatic condition was sharply reduced through the use of the magnets; also that the magnet will impede the migration of a foreign object into a vital organ if penetration should occur.

Medullary Nailing of Fractures

Medullary nailing may be indicated in simple transverse or short oblique fractures of the middle third of the long bone, but it is not recommended for routine use since it requires an open reduction (often converting a simple fracture into a compound one with added dangers of infection) and because of the special equipment required.—*J.Am.M.A., July 23, 1955.*

Platelets, tiny cell fragments found in the blood, arrest bleeding by sticking to the edge of the wound and to each other until they pile up into a little cork that fills the hole.—*Sci. News Letter, May 14, 1955.*

Predicting the weight-gaining ability of a young bull and of his offspring by determining his heart rate is being attempted by scientists at Oregon State College. A correlation between the two was found. —*Farm J., Sept., 1954.*

Anatomical Conditions in the Dog that Favor Dermatitis

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When considering a case of canine dermatitis, it is well to bear in mind any anatomical abnormalities which may predispose to skin irritation.

There are at least five such abnormalities: (1) Around the mouth, particularly in Spaniel-type dogs, there is, on the lateral surface of the lower lips midway between the commissure and the canine teeth, a fold of skin that forms a recess which is continually moist from the saliva of the mouth. This harbors bacteria and fungi which often cause the area to be ulcerative and to have an objectionable odor. (2) The external ear canal does not have natural drainage and furthermore, in dogs with hanging ears, it is difficult to ventilate the ear canal. Bacteria and fungi collect here and may cause an ulcerative type of infection that produces irritation and an offensive odor. (3) The anal sacs are also difficult to drain. When infected and irritated, the animal, by licking with the tongue, may inoculate other parts of the body with the bacteria and fungi. (4) In fat females, where the perineal skin meets the skin of the vulva, it forms a crease which has poor drainage and ventilation, particularly in spayed bitches where atrophy of the vaginal tract occurs. (5) In the cryptorchid male, the internal secretion of the undescended testicle often disturbs the hormonal balance of the dog, particularly in those animals where the testicle undergoes tumor formation of the Sertoli type which produce an excessive amount of estrogen. This results in alopecia, loss of libido, and feminization with attraction of other male dogs. There is elongation and swelling of the penile sheath, hypertrophy of the mammary glands with a lengthening of the nipples, and sometimes mammary tumors develop. There is also pigmentation and thickening of the skin on the abdomen and scrotum.

Each of these anatomical defects can be corrected surgically: (1) The recesses or folds of the lips can be excised and the edges of the skin sutured. (2) The ear

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canal can be drained surgically by the method used by the Angell Memorial Animal Hospital.[1] (3) The removal of the anal sacs as prescribed by Theobald[2] has done much to relieve infected anal sacs. (4) The removal of a horseshoe-shaped portion of the skin lateral and dorsal to the vulva lifts the latter and removes the crease.[3] (5) Removal of cryptorchid testicles has proved highly satisfactory in restoring normal endocrine balance in dogs so affected.

In summary, it has been pointed out that there are at least five anatomical peculiarities in certain dogs which predispose to skin irritations. Surgical procedures for the correction of each is worthy of consideration.

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[2]Theobald, A. R.: Surgery of the Anal Sacs. North Am. Vet., *23*, (1942): 44-47.

[3]Blakely, C. L.: Surgical Correction of Vulvan Dermatitis, in "Canine Surgery." 3rd ed. American Veterinary Publications, Evanston, Ill. (1952): 475-478.

Estrogens in Pasture Plants

Subterranean clover from all districts of New Zealand, and in all seasons, had a high estrogen content; white clover had practically none. Red clovers had an appreciable content and, in the early spring, dry grass had a low but definite estrogen content. No breeding problems in sheep have been associated with this estrogen in New Zealand but it has caused lactation in wethers.—*Vet Bull., July, 1955.*

Rate of Bovine Fetal Growth.—The calf which weighs 85 lb. at birth, as a fetus at 2 months weighed 3 oz. (the size of a mouse); at 3 months, 8 oz.; at 4 months, 2.5 lb.; and at 6 months, 18.1 lb.—*Hoard's Dairyman, June, 25, 1955.*

This photograph, submitted by Dr. Philip C. Clinger, Rochester, Ind., shows a 7-year-old cow with her 2 calves which were born three weeks apart, the darker one (in the foreground) on August 17, the other on Sept. 9, 1954. The cow's teats were reported to have "waxed" and the milk to have retained a colostral character until after the second calf was born. When examined rectally three months after the last calving, her uterus seemed normal with the right horn containing a single 2-month fetus. Previously she had given birth to 4 normal calves.

Poulardization of Native Ducks

JOSE B. ARAÑEZ, D.V.M., and CONRADO S. SAGUIN, D.V.M.

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POULARDIZATION is the surgical removal of the ovary in birds. This operation has long been known in poultry and, like the caponizing of males, is undertaken to hasten the growth and improve the quality of the flesh. However, poulardization of ducks or other female birds is rarely done, probably because of the intricate operative procedure involved.

The production of eggs is the main purpose of duck raising in the Philippines. However, bigger profits may be obtained by the duck raisers if poulardization of culled young ducks is practiced.

As far as the writers are aware, no work on poulardization of ducks has yet been reported or done.

MATERIALS AND METHODS

Thirty-five 2-month-old native ducks, each identified by a numbered legband, were used.

The essential surgical and laboratory equipment used were: scalpels, scissors, caponizing scoop, spreader, pointed probe, weighted cords, forceps, drape, cotton, suturing needle, linen thread, and syringe.

The drugs used were: sulfanilamide powder, ethyl alcohol, tincture of iodine, chloroform, and 5 per cent dextrose in saline solution.

One large cage 7½ by 3 by 1 ft., 15 small cages 1½ by 1 by 1 ft., and a small shed 10 by 6 by 5½ ft. provided with a divided wire enclosure 19 by 10 by 2½ ft. were used in housing the ducks.

Preoperative Care, Feeding, and Restraint.—Before the operations were performed, a study of the surgical anatomy of the gonads and the operative area was conducted by dissecting 2 chloroform-killed 2-month-old ducks. Eight ducks, ranging from 1 to 4 months old, were used for practice operations.

The ducks were given proper care and observation in cages for five consecutive days before the operation. Microscopic examinations of the droppings were made frequently to detect the presence of parasites. The birds were fed a growing mash consisting of 4 parts rice bran, 2 parts ground corn, 2 parts copra meal, 2 parts fish meal, 1 per cent sodium chloride, and 2 per cent ground shell, morning and afternoon. Supplementary feeds such as palay, corn, and chopped green grass were given during the intervals of regular feeding times. The feeds were placed in a long wooden trough to avoid overcrowding during feeding.

The ducks were divided into two groups: principals and controls. The principals were fasted 24 hours prior to the operation. The wings of the bird were secured by a cord looped around the base of the wings. The free end of the cord was provided with a weight which was allowed to hang. The same procedure was followed to restrain the legs (fig. 1).

Operative Technique.—The feathers at the site of operation, on the left side of the body, were plucked. The skin was disinfected with tincture of iodine, then de-

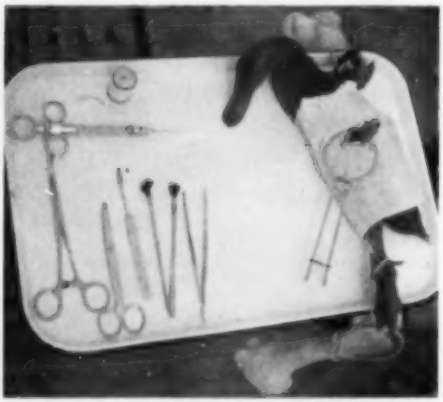

Fig. 1—A properly restrained and draped 2-month-old native duck with a caponizing spreader in place. The equipment and instruments used in the operation are also shown.

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This is a portion of an undergraduate thesis presented by the junior author on March 24, 1955, in partial fulfillment of the requirements for the degree of doctor of veterinary medicine.

This work was done from June 15, 1954, to Feb. 2, 1955, under the direction of the senior author.

The authors gratefully acknowledge the help of Dr. L. M. Yutuc, head of the Department of Veterinary Medicine and Surgery, and the suggestions of Dr. J. A. Solis, head of the Department of Veterinary Anatomy from the same college.

colorized with ethyl alcohol, and a clean drape was applied. The intercostal space between the sixth and seventh ribs was palpated by the left hand and the skin over it was drawn and held back toward the hip. Using the right hand, an incision ½ to 1 inch long was made through the skin. The sartorius muscle was then pushed backward and the intercostal space punctured, the thigh muscles being held back by the left hand. Care was observed dorsally not to injure the left kidney, and the incision was made close to the anterior border of the seventh rib to avoid the intercostal artery.[1] After incising the intercostal muscles, the spreader was inserted between the ribs (fig. 1). The peritoneal membranes were next punctured and torn off by a small hook-pointed probe. The intestines were pushed down and back, and the ovary was exposed. It is attached to the dorsal wall of the abdomen and covered by a membrane which was torn off with a hook-pointed probe leaving the ovary bare. The caponizing scoop was applied to the base of the ovary and with several twisting movements it was finally severed from its attachment without injuring the posterior vena cava which lies directly dorsal to it. The slight hemorrhage which usually occurred was easily controlled by applying pledgets of absorbent cotton. Sulfanilamide powder was dusted at the site of the enucleated ovary. When the rib spreader and the drape were removed, the skin and thigh muscles automatically returned to their natural position to cover the incision. The skin incision was then closed by three interrupted linen sutures. The right side of each duck was also opened to determine the absence or presence of the right ovary. The ablated ovary was weighed and its weight recorded.

Postoperative Care and Feeding.—Immediately after the operation, the bird was given 25 cc. of 5 per cent dextrose in saline solution into the pectoral muscles. Food was withheld for 24 hours. The wound was observed and precautions taken to prevent the development of wind puffs. On the sixth day, the sutures were removed.

On the twelfth week, the ration was changed to laying mash consisting of 4 parts rice bran, 3 parts copra meal, 2 parts ground corn, 1 part fish meal, 1 per cent sodium chloride, and 2 per cent ground shell, until the end of the experiment.

Fig. 2—A poulardized and an unpoulardized native duck 28 weeks after the experiment.

Troughs were provided containing enough water for them to swim. Sanitary measures were maintained. The weekly weight of each bird was taken and recorded.

Behavior and Cooking Experiment.—Any difference in the behavior and development of the poulardized and unpoulardized birds was carefully recorded.

All the poulardized and 6 control ducks were killed after the twenty-eighth week. Two from each group were dressed and cooked with the same ingredients, process, and length of time and the taste, odor, and palatability were observed.

DISCUSSION

Operative Site and Anatomy of the Gonads.—The incision between the sixth and seventh ribs is the same as for the caponization of native drakes.[2] An eighth, thin and undeveloped rib is present.

The right ovary and oviduct were absent in all of the ducks poulardized, the same as in chickens.[3] The left ovary appears as a group of loosely joined, vascular, yellowish, and spherical bodies representing the

Fig. 3—Two dressed poulardized (right) and 2 unpoulardized (left) native ducks 28 weeks after the experiment.

ova. It is located slightly to the left of the median plane in the lumbar region and is attached to the dorsal wall. It is related laterally to the left lung and internal intercostal muscles; ventrally, to the proventriculus, spleen, and small intestine; anteriorly, to the membranous diaphragm which separates it from the left lung; and dorsally, to the anterior lobe of the left kidney and to the posterior vena cava, to which it is partly adherent at the junction of the right and left common iliac veins. The aorta is immediately dorsal to these veins. The ovary extends from the level of the fifth to the eighth ribs. The oviduct extends caudally against the dorsal part of the left body wall to which it is attached by a narrow fold of peritoneum. It runs posteriorly in relation to the ilium and ischium, emptying into the cloaca lateral to the opening of the left ureter.

Proper Age and Size to Poulardize.—After a series of trials, ducks 2 months old and weighing about 400 Gm. were preferred because the ovary and blood vessels were still small and death due to hemorrhage was less likely to occur. Furthermore, only a small surgical incision was necessary.

Table 1 shows the time required for the operation and for healing, the weight of the removed ovary, and postoperative complication. The average time consumed per operation was 24.6 minutes. This can be reduced as the operator becomes more experienced and if only the left side is opened.

The weights of the removed ovaries ranged from 0.2 to 0.6 Gm. with an average of 0.3 Gm., and the duration of healing ranged from six to eight days. All the surgical wounds healed by first intention. Wind puffs, caused by air trapped underneath the skin near the wound, developed in 13.33 per cent of the birds. This condition was probably minimized by the skin suturing. It is easily remedied by puncturing the skin over the area with a 15-gauge needle or by cutting a V-shaped incision in the skin. No deaths occurred.

Graph 1—This shows the average weekly gain in weight per duck of the 15, 2-month-old poulardized and 15, 2-month-old unpoulardized native ducks.

TABLE 1—Showing the Duration of Poulardization, Weight of the Removed Ovary, Duration of Healing, and Postoperative Complication of 15, 2-Month-Old Native Ducks

Duck (No.)	Duration of poulardization (minutes)	Weight of removed ovary (Gm.)	Duration of healing (days)	Postoperative complication
1	20	0.6	7	None
2	35	0.3	6	None
3	30	0.5	6	Wind puffs
4	26	0.5	8	None
5	25	0.3	7	None
6	30	0.2	6	Wind puffs
7	22	0.2	6	None
8	25	0.2	7	None
9	23	0.2	7	None
10	19	0.4	8	None
11	21	0.2	7	None
12	24	0.3	7	None
13	22	0.2	6	None
14	23	0.2	8	None
15	24	0.2	6	None
Ave.	24.60	0.3	6.80	13.33%

Gain in Weight.—During the first four weeks, except the third, the control ducks showed a higher average weekly gain in weight than the poulards (graph 1). From the fifth week until the end of the experiment, the poulards consistently had the greater average weekly gain. The abrupt decline in weight observed on the fifteenth week (graph 1) coincided with a storm that continued for almost five days. The maximum average weekly gain among the poulardized group was 94.0 Gm. per bird at the twentieth week, after which the weekly gain in weight declined. In the control group, the maximum weekly gain was an average of 81.92 Gm. per bird in the twenty-third week. The poularized ducks had gained 223.96 Gm. per bird more than the controls by the twenty-eighth week (fig. 2 and 3).

The poulards appeared lazy, docile, quiet, and easier to handle than the unpoulardized ducks. Their feathering was thicker and longer. They had rounded chests, yellowish skin, and ample fat deposits, while the unpoulardized ducks were thin and had whitish skin. There were no "slips."

The cooking time for the poulards was much shorter; 72 minutes, compared with 105 minutes for the control birds. The "ducky" odor was practically eliminated, and the cooked flesh of the poulards was more palatable and tender.

SUMMARY

1) Native ducks are best poularized when 2 months old while the ovary is still small and there is less danger of death due to hemorrhage.

2) The appropriate site of incision is between the sixth and the seventh ribs.

3) The right ovary and oviduct were absent in all the ducks poularized.

4) No postoperative complication other than wind puffs was observed.

5) The poulards made the greater gain in weight, the highest gain being in the twentieth week; thereafter, a gradual decline was noted.

6) It is most economical to market the poulards from the twentieth to the twenty-first week after poulardization, before the gains begin to decrease.

7) The poulards fattened more easily, were easier to handle, and their feather growth was faster.

8) The characteristic "ducky" odor of the flesh was apparently eliminated by poulardization.

9) Poulard flesh seemed more palatable and soft, and required less time to cook than that of the control birds.

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Foreign Body in the Esophagus of a Horse

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A Percheron mare, 17 years old, when seen on Jan. 5, 1953, had a large palpable mass, 10 cm. in diameter, just posterior to the mandible in the region of the esophagus. When a stomach tube would not pass the object, the mare was referred to the veterinary clinic at the University of Illinois.[*]

All attempts to pass the stomach tube were unsuccessful. The mare sneezed at frequent intervals and, when she attempted to drink, the water was regurgitated through the nose. Since she resented any attempts at examination by way of the mouth, she was given 1 liter of saline solution intravenously and allowed to rest.

The following day, further attempts to pass the stomach tube were still unsuccessful and the mare again refused to allow an examination by way of the mouth. Radiographs then made of the area showed the presence of a V-shaped piece of wire lodged in the anterior part of the esophagus (fig. 1). The mare was prepared for surgery, secured on the operating table, and anesthetized with 450 cc. of equitol given intravenously. Attempts were made to explore the pharynx and esophagus but the hand of the operator was too large. However, a student with a small hand was

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*The case was referred by Dr. Richard Dye, Canton, Ill.

able to reach the foreign body by extending his fingers through the pharynx and into the esophagus. After several unsuccessful attempts with uterine forceps, wire hooks, bronchoscope, and a canine gastroscope, he was able to secure the foreign

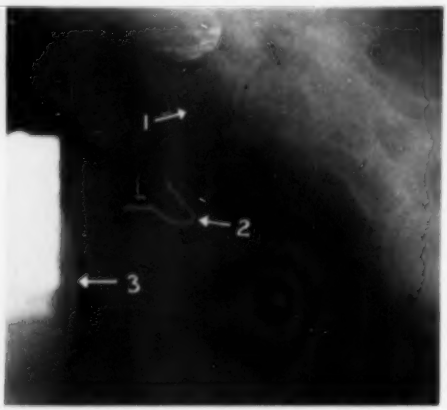

Fig. 1—Axis vertebra (1); foreign body (2); and angle of the mandible (3).

body with his fingers and remove it. The V-shaped wire was 3 inches long. It was lodged with the apex pointing toward the stomach and with the ends imbedded in the wall of the esophagus.

After removal of the foreign body, a stomach tube was passed readily and fluids, dextrose, and milk were given. A liter of saline and dextrose solution was also administered intravenously.

An extensive edematous swelling soon developed in the area where the foreign body had been lodged. Antibiotics were administered and short-wave diathermy was applied daily. The swelling gradually subsided in the next three days. Saline, saline-dextrose, and aminosol were administered intravenously for several days and although the mare developed a severe diarrhea during her convalescent period, she was fully recovered when discharged on January 22.

Surgery is indicated in pulmonary hydatidosis. Resection of the cysts in 258 persons resulted in postoperative complications in 12.8 per cent and death in 2.1 per cent of the patients.—*J.Am.M.A., July 16, 1955.*

The Evaluation of Bull Semen

A study was made on the value of certain characteristics of bull semen for estimating its fertilization capacity (Marcus W. H. Bishop and J. L. Hancock in *Vet. Rec.*, May 14, 1955). The following measurements were studied: volume of ejaculate, concentration of spermatozoa, incidence of dead or morphologically abnormal spermatozoa, resistance of spermatozoa to temperature shock, concentration of fructose, methylene blue reduction, fructolysis, oxygen uptake, motility, and impedance change frequency (I.C.F.), an electric induction test.

The mean values for measurements of these characteristics in 121 semen samples from 76 bulls are shown in a table. The article states that: "The large amount of variation encountered in most of [these] . . . is . . . very striking . . . ."

With reference to the "interrelationships of semen characteristics," it states.

> . . . it is convenient to distinguish fertilizing capacity from other semen characteristics, . . . referred to as 'test characteristics.'
>
> The information obtained from a study of the interrelationships of the test characteristics is of practical interest because it enables one to see if the different tests measure different characteristics or if they each individually tend to give the same sort of information. Obviously if two tests measure the same characteristic one is at liberty to retain the more accurate or . . . the more convenient.

The test characteristics have been collected into five groups as follows:

1) Ejaculate volume, concentration of spermatozoa and concentration of fructose.
2) The incidence of abnormal spermatozoa.
3) The metabolic activity of spermatozoa (methylene-blue reduction, fructolysis and O_2 uptake).
4) The physical activity of spermatozoa (motility and I.C.F.).
5) The resistance of spermatozoa to temperature shock.

These interrelationships are shown diagrammatically. The article concludes:

> The results of this survey suggest that the most useful indices of the fertilizing capacity . . . are measurements of the number and percentage of living spermatozoa present, and their degree of physical activity. This information can be conveniently obtained by measurement of the concentration of spermatozoa, the incidence of dead spermatozoa and I.C.F. Information obtained from morphological studies may be valuable because it is generally not accessible by other tests and because in special cases it can have a very clear bearing on fertility.

One of the great advantages which the British have over American veterinarians is that the authors of papers are given adequate time to present their material and, of greater importance, they are given the opportunity for full discussion by contemporary workers.

For example, in the discussion following the presentation of this paper, Mr. G. F. Smith states:

> We have been presented with one of the most helpful pieces of work on bovine semen so far undertaken in this country, and in fact to the best of my knowledge, in the world. . . . We are now in a position to appreciate the advantage of the impedance bridge in fertility estimation of semen, for a quantitative test to interpret the motility of samples which is not dependent upon the individual's eye has been long overdue.—*H. E. Kingman, Sr., D.V.M., Georgia.*

Rupture of the Uterus in a Cow

An Ayrshire cow, 4 years old and due to calve, had shown signs of labor three days previously but subsequently had seemed content. However, she had stopped eating and her eyes were sunken. When brought to the clinic, her temperature, pulse, and bowel movements were normal but rumen peristalsis was absent. Upon vaginal examination, the cervix was dilated, the fetal membranes could be felt, and the hand came in contact with intestines and other organs. Not until two men pushed the ventral wall of the cow's abdomen upward could the fetus be reached. The calf was still alive.

Laparotomy, using paravertebral anesthesia, was performed through the upper right flank with the cow in a standing position. The umbilical cord became entangled and ruptured so the calf was dead on delivery. The right horn of the uterus had an 18-inch laceration extending along its lateral aspect from the apex to the body of the uterus. It had everted due to contraction of the muscle. The membrane was removed, and the uterus was closed, with some difficulty, with two lines of a Connell suture, using No. 2 chromic catgut. There was a considerable quantity of sanguineous exudate in the peritoneal cavity but no evidence of peritonitis.

After the operation was completed, the cow was given 50 mg. of stilbestrol dipropionate, 10 ml. of pituitrin (repeated in 12 hours), 3 million units of procaine penicillin, and 500 I.U. of tetanus antitoxin. She made a remarkable recovery and milked well but did not again become pregnant.—*Austral. Vet. J., May, 1955.*

[Interesting comments by the authors (B. R. Hutchings and D. C. Blood of the veterinary faculty, University of Sydney) include the following: 1) A history of labor, then cessation usually indicates an open cervix with no fetal parts in the vagina; (2) entry of the fetus into the posterior part of the genital tract seems necessary to stimulate labor; (3) the cow's recumbency and lack of ruminal movement, since the inception of labor, probably indicated abdominal discomfort but there was no inflammation; (4) recession of the eyeball was probably due to dehydration from the continued loss of fluid into the peritoneal cavity; and (5) cows are not uncommonly infertile following cesarean section.]

Effect of Fed Hormones on Swine

Methyltestosterone, 20 mg. per animal daily, or diethylstilbestrol, 2 mg. per animal daily, with and without terramycin,® did not improve the growth rate or feed efficiency of swine in drylot. The testosterone increased masculine characteristics and increased the lean meat by 5 per cent. The stilbestrol stimulated the mammary growth in both sexes and the enlargement and congestion of the vulva in the females, but it had less effect on the lean meat content of the carcass.—*J. Anim. Sci., May, 1955.*

Immobilizing Turkeys for Slaughter.—Turkeys can be anesthetized effectively with carbon dioxide gas, thus reducing the struggling and resultant bruising during slaughtering.—*U.S.D.A.*

A Rare Neoplasm in a Cow's Uterus

A 16-year-old cow with normal estrous periods, but which failed to conceive for four years, was found on necropsy to have a uterus measuring 36 by 18 inches. The lumen was intact but the uterine wall was solid white tissue of a somewhat rubbery consistency with a few pale yellow areas. The lymph nodes and other organs were not involved. Histologically, the tissue consisted of a relatively acellular and avascular network of collagen fibers with a few scattered bundles of muscle fibers. The diagnosis was neurofibrosarcoma.—*Austral. Vet. J., March, 1955.*

CLINICAL DATA

Preliminary Results in the Control and Treatment of Shipping Fever Complex in Beef Cattle

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Wooster, Ohio

MANY ATTEMPTS to develop means for the control of so-called "shipping fever" have been reported. It would serve no worthwhile purpose to review at this time the early efforts aimed at the development of protective measures against the disease, other than to state that no prophylactic treatments have been developed which have been shown to produce outstanding success in the control of shipping fever.

The prophylactic inefficiency of all biological products has become more evident since the disease, or a disease resembling shipping fever, seems to have become more virulent in recent years. This observation may be due to the fact that in recent years there is a tendency on the part of beef cattle feeders and breeders to buy younger animals or those that may be more susceptible to shipping fever.

The inadequacy of bacterins, aggressins, and serums when given to cattle following shipment is well documented in veterinary literature. Scott and Farley[1] in 1932 investigated thousands of cattle vaccinated with bacterins or aggressins at the Kansas City Stockyards and found little or no benefit from the use of these biological products in preventing so-called shipping fever.

Palotay[2] in 1953 reported that the use of anti-hemorrhagic septicemia serum, when administered to beef cattle on arrival at feedlots, was not economically justified. Of 667 animals given this serum, 180 (27%) were brought back for treatment, while of the 660 controls only 21.8 per cent were returned for treatment.

Graham[3] in February of 1954 reported that he had encountered a disease in cattle associated with a Pasteurella infection in which the antibiotics, sulfonamides, and antiserums had little or no effect on the course of the disease.

Carter[4] examined 26 cases of typical shipping fever in cattle ranging in age from 4 to 12 months. The principal pathological process observed was an acute bilateral bronchopneumonia. He concluded that the disease as it appeared in central Canada was a true pasteurellosis.

Today, most veterinarians are relying on various antibiotics and sulfonamides for prevention and treatment, rather than the use of the products prepared from Pasteurella infections.

Without a better understanding of the cause of shipping fever, the prevention and treatment of the disease likely will continue to be ineffective. This, then, serves to emphasize the need for detailed study of the disease in an attempt to learn more about the cause and thereby attempt to improve the methods of prevention and treatment.

PROPHYLACTIC TREATMENT ON ARRIVAL—1952

During the fall of 1952, the Department of Veterinary Science of the Ohio Agricultural Experiment Station, in cooperation with workers in the Department of Animal Science,[5-7] began to investigate the use of some preventive and therapeutic measures for controlling shipping fever. At this time, two separate lots of feeder cattle obtained for use in breeding and feeding experiments were received from western shipping points.

One shipment consisted of 134 calves that averaged 452.8 lb. at Kansas City. These animals were shipped on October 7 and arrived at Wooster, Ohio, October 10. After unloading, the cattle were trucked 2 miles to the Station's beef feeding barn. Four watering tanks, one to each of two pens, were provided.

Sixty-two calves picked at random were given 40 cc. of anti-hemorrhagic septicemia serum subcutaneously before the cattle were put in their respective pens.

Two pens of 18 calves were given sodium acetate and two pens, sodium propionate, in their drinking water in an attempt to

From the Ohio Agricultural Experiment Station, Wooster.
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supply energy-yielding compounds orally. It was estimated that each steer received 75 Gm. of the compound daily for three days, assuming that 4 gallons of the mixture was consumed each day. In two additional pens, sodium bicarbonate was added to the water at the same estimated rate, but for one day only.

TABLE 1—Prophylactic Treatment of 134 Feeder Calves

Pens	No. of animals	Treatment at unloading	
1 and 2	36	Sodium propionate in drinking water	18 head were given 40 cc. serum.
3 and 4	36	Sodium acetate in drinking water	18 head were given 40 cc. serum.
5 and 6	36	Sodium bicarbonate in drinking water	18 head were given 40 cc. serum.
7 and 8	26	Water only	8 head were given 40 cc. serum.

Nothing was added to the water in the remaining two pens. There was no noticeable difference in the water intake between the various pens. Serum-treated and untreated control animals were included in each of the previously described pens (table 1). All cattle were fed mixed hay only for the first thirty days after which grain was included in the ration.

All of the 134 calves appeared tired on arrival. There was some coughing and a few calves did not eat. Symptoms of shipping fever—labored breathing, diarrhea, depression, loss of appetite, and elevated temperatures which frequently reached 107 F.—were observed to develop between the second and seventh day after arrival. Approximately 14 per cent of the cattle required treatment. Shipping fever or a shipping fever-like disease occurred in all groups irrespective of the prophylactic treatment received.

Thus it appeared that the anti-hemorrhagic septicemia serum administered on arrival was of little or no value in preventing the disease. Likewise, acetate or propionate feeding at the level administered did not reduce the incidence of the disease.

Sulfathiazole or sulfamethazine orally to clinically ill animals appeared to be a satisfactory treatment.

One calf died two days after arrival. Necropsy showed the presence of a large quantity of fluid in the chest cavity and complete consolidation of both lungs.

In addition to the group of 134 calves received October 10, another shipment of 50 bull calves weighing 400 lb. was received and placed in a different barn approximately ¼ mile from the beef feeding barn. This group was shipped directly from a ranch in Texas. Sixteen animals (32%) in this group required therapy for shipping fever. These calves responded rapidly to sulfonamide medication. It appeared that a less virulent strain of organism, or less severe conditions of transportation and exposure, or both, may have accounted for the moderate severity of the disease.

This conclusion was partly responsible for the decision to buy cattle direct from the ranch for breeding and feeding investigations for the following year.

PROPHYLACTIC TREATMENT PRIOR TO SHIPMENT—1953

In the fall of 1953, 248 Hereford calves weighing 450 to 550 lb. were purchased and shipped directly from two ranches in northwestern Texas to the Ohio station. These calves, as well as their dams, were in excellent condition at the time of shipment. The handling of the calves at the time of segregation, treatment, and loading for shipment was done with utmost care. Railroad cars used in the shipping of these calves were clean and bedded with fresh sand.

Calves were left on dams but taken off water approximately 12 hours before they were weighed. Following weighing, all animals were treated for ear ticks and given preventive treatment for shipping fever (table 2).

Of the 248 head of cattle shipped, 73 (30%) picked at random, received 1,500,000 units of the long-action type of penicillin (bicillin*) intramuscularly. Another 73, also picked at random, received 50 cc. of anti-Corynebacterium-Pasteurella serum subcutaneously, and 31 purebred heifers and 2 purebred bulls were treated with both penicillin and serum. To serve as controls, another 69 (30%) animals, also picked at random, were left untreated. All treated groups, with the exception of the purebred animals, were divided equally among the five cars.

*Bicillin was supplied through the courtesy of the Wyeth Laboratories, Inc., Philadelphia, Pa.

One car containing 46 calves was loaded Sept. 28, 1953, at 6:30 p.m. This car was scheduled to leave the yards at 9:00 p.m. The shipment arrived in Wooster six days later at 6:30 p.m.

Four cars containing 202 calves were loaded on Sept. 30, 1953, at 3:30 p.m. The cars moved from the yards at 5:30 p.m. One of us (N.B.K.) accompanied this shipment in order to make observations on the feeding, watering, exposure, and general management of the cattle from the shipping point to the final destination in Wooster.

TABLE 2—Preventive Treatment Prior to Shipment

Car	Penicillin	Antihemorrhagic septicemia serum	Serum and penicillin	No treatment	Totals
A	17	17	....	16	50
B	17	17	....	16	50
C	17	17	....	16	50
D	17	17	2	16	52
E	5	5	31	5	46
Total	73	73	33	69	248

As a rule, the carloads of cattle were positioned near the engine in the train to make them more comfortable by reducing the slack-action. Usually, about 80 to 100 cars were in the train. Other cars of cattle were also included on this train. The only regular stops made were at the division points, approximately every 110 miles. At these points, there was usually a delay of 45 minutes to as long as six hours. Usually this delay was due to changing of train crews, servicing of equipment, and switching of cars on the train. It was these long, seemingly avoidable delays which accounted for approximately one-half or more of the total time necessary to complete the trip.

Approximately 33½ hours were required to reach the sixth division point and rest station at Fort Madison, Iowa, which was approximately 650 miles from the shipping point. The cattle were unloaded at 3:00 a.m. on October 2 and each car load was placed in separate pens. Water and 200 lb. of prairie hay were given to each pen of cattle. The cattle drank less than the normal amount of water and each carload ate approximately 100 lb. of the hay. Watering and feeding facilities were inadequate due to leaky troughs and lack of feeder space. A constant supply of water was not maintained in spite of the high (100 F.) temperatures during the daytime. The yards were partly covered, but were dusty and there was very little bedding. The cattle were uneasy, milling around the pens most of the time.

The cattle had an opportunity for disease exposure because the disinfection and cleaning between handling of different groups was usually inadequate. Also, there was opportunity for contact with animals in adjacent pens. It appeared that much could be done to improve not only facilities for handling cattle but also measures to reduce unnecessary delays all along the road.

The four cars of cattle had been en route three days when they arrived at their final destination on October 3. All the animals were unloaded at the same time. The steers were placed in the beef feeding barn and the bulls in another barn approximately ¼ mile away. All animals were weighed at the time of unloading. The average weight loss was found to be approximately 53 lb. per animal. Each carload of animals was maintained as a unit. Car A became lot 1, car B, lot 2, etc.

On arrival, all animals appeared to be in good condition but were tired and uneasy. In all groups, regardless of treatment, temperatures ranged from normal to 105 F., averaging approximately 103 F., and feces were firm and contained some mucus. Body temperatures of representive animals, taken each day for the first week after arrival, ranged from normal to slightly elevated regardless of prophylactic treatment. During the first week, coughing and some diarrhea were observed in most of the cattle.

It was the plan of the experiment that no treatment either prophylactic or therapeutic would be given at Wooster until such measures became necessary in an attempt to save the animal.

The cattle had a fair quality of timothy-mixed clover hay before them at all times.

Beginning on October 7 and continuing for three weeks, daily observations were made to determine the number and treatment received by the animals refusing to eat at time of putting fresh hay in the feed bunks. There was no difference between treatment groups in this respect. While it was apparent that the bunk space was not sufficient to enable all animals to eat at one time, it was thought that this observation might be used as a yardstick in ascertaining the value, if any, of the prophylactic treatment.

On October 9, six days following arrival, a steer from the penicillin-treated group in lot 2 was the first to require treatment for shipping fever. He had a temperature of 105.0 F., nasal discharge, and rapid breathing. On October 11, a control steer had a temperature of 106.2 F., nasal discharge, depression, dehydration, and slight diarrhea. Both of these animals were treated with sulfathiazole intravenously and orally.

On October 19, all steers were injected with tuberculin and on October 20, the bulls were injected with tuberculin and blood samples were collected for brucellosis testing. The test readings were made three days following injection of the tuberculin. Bulls were stanchioned for this work while steers were merely crowded into the feedway.

During the six weeks, from their arrival until November 15, approximately three fourths of the 248 cattle in this experiment exhibited one or more of the following clinical signs: elevated temperature (103 to 108 F.), chills, anorexia, dyspnea marked by extended head, open mouth and protruded tongue, "pumping" respiration, coughing, diarrhea, ocular and nasal discharges.

Of the 248 cattle, 71 (29%) were treated therapeutically or required treatment in addition to the prophylactic treatment given before shipping. Of

those given penicillin and serum simultaneously 15 per cent were re-treated. Of the penicillin group, 20 per cent were re-treated, while of the serum-treated group, 29 per cent required re-treatment. Of the control group, 43 per cent were treated for the disease (table 3).

TABLE 3—Effectiveness and Statistical Significance of Various Prophylactic Treatments for Control of Shipping Fever

Prophylactic treatment at time of shipping	Animals requiring treatment (No.)	(%)	Significance of effectiveness	Deaths due to shipping fever
Penicillin only	(73) 15	(20)	P 0.01	2
Serum only	(73) 21	(29)	Not sig. P 0.10	3
Controls no treatment	(69) 30	(43)	.....	5
Penicillin and serum simultaneously	(33) 5	(15)	P 0.01	1
Totals	248 71	(29)	.....	9 (3.6%

When compared with the control group, all treatment groups showed a lower incidence of the disease. The results with serum alone did not reach the customary 5 per cent level of significance. The results with penicillin alone and in combination with serum were significant at the 1 per cent level.

At the start of the outbreak, the animals were treated therapeutically with sulfathiazole intravenously and orally; later, the treatment was changed to triple sulfonamides, orally, only. This change was made due to the observation that the restraint necessary for giving intravenous injections was detrimental. Some animals were treated with aureomycin® and terramycin.® These drugs did not seem to be any more effective than the sulfonamides. Supportive therapy in the form of dextrose, eggs, water, and rumen transfusions was instituted in some instances.

During this acute outbreak, 9 animals died. All were found with the following lesions at postmortem: bilateral pneumonia with extensive consolidation and emphysema; trachea and bronchi extremely congested and hemorrhagic; subcutaneous edema of neck and throat; lymph nodes in throat and neck region swollen and hemorrhagic; fibrinous pleuritis was also present. The deaths were essentially evenly divided among the control and treatment groups.

Bacteria (*Pasteurella multocida*), usually associated with shipping fever, were isolated from the lung tissue of 8 of the 9 dead animals Lung, liver, and spleen tissues taken from the dead animals were frozen and further studies are being carried on to determine if other agents may have been involved in this condition.

CONCLUSIONS

No final conclusions have been reached concerning the results of these tests. It would seem, however, that under the conditions of these experiments anti-hemorrhagic septicemia serum used prophylactically on cattle following shipment had little or no value in controlling shipping fever or a disease resembling shipping fever. Likewise, sodium acetate and sodium propionate feeding, at the level administered, did not reduce the incidence of the disease.

While a larger percentage of the controls required therapeutic treatment, nevertheless, neither penicillin nor serum when used prophylactically prior to shipment gave satisfactory protection.

In light of the conditions encountered, a question might logically be raised as to the need for livestock disease control officials and railroad officials to study measures for improving the sanitation and facilities in caring for cattle in transit. In addition, much could be done to reduce shipping fatigue by eliminating unnecessary delays in total shipment time.

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Studies on Ketosis in Dairy Cattle. XIX. Glucocorticoid and ACTH Therapy in the Los Angeles Area

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PREVIOUS WORK in the Maryland area has demonstrated the effectiveness of cortisone acetate, ACTH, hydrocortisone, and a combination of hydrocortisone and hydrocortisone acetate[1-3] for the treatment of bovine ketosis. In these studies, it was observed that 1.5 Gm. of cortisone acetate or 200 to 600 A.V.U. (Armour veterinary units) of ACTH in gel were sufficient for most cases, but that larger doses resulted in even more rapid recovery. An intramuscular injection of 1.0 Gm. of either hydrocortisone, or a combination of equal parts of hydrocortisone and hydrocortisone acetate, was usually adequate. Hydrocortisone was much more effective than hydrocortisone acetate evaluated in terms of clinical response, increase in blood glucose, and disappearance of urine ketones. The combination appeared to offer some promise since the alcohol form (hydrocortisone) was rapid in action, whereas the acetate form appeared to be slower in action, the blood glucose increasing gradually over a period of days.

The objective of the present study was to establish the minimum and optimum dosage of hydrocortisone alone and in combination with hydrocortisone acetate. An additional objective was to establish the value of a relatively small amount of hydrocortisone when given in conjunction with glucose. The study was conducted in the Los Angeles area at the suggestion of one of the authors (C.H.O.) because of the relatively large number of cases within a small area and the fact that a large percentage of cows in this area did not exhibit a satisfactory response to the usual methods of treatment.

EXPERIMENTAL STUDIES

Ninety-seven cows, diagnosed as having primary ketosis, were treated with varying dosages of hydrocortisone, hydrocortisone and hydrocortisone acetate (1:1), ACTH in gel, or cortisone acetate. Of these, 61 received a single dosage of a total of 0.5 to 1.0 Gm. of hydrocortisone. Of the cows which received 1.0 Gm. or less of hydrocortisone or the combination of hydrocortisone and hydrocortisone acetate, 25 received a simultaneous intravenous injection of approximately 150.0 Gm. of glucose and 8.5 Gm. of calcium as calcium gluconate or calcium chloride. Seven cows received 1.0 Gm. of the combination of the two forms of hydrocortisone, followed by 0.5 Gm. on each of the next two days. Five cows received 1.5 Gm. of the combination of hydrocortisone followed by 1.0 Gm. on the following day. In order to make a comparison with results obtained in the Maryland area, a number of cows were treated with ACTH, and 1 received relatively large dosages of cortisone acetate. In all cases, the glucocorticoids and ACTH were administered by intramuscular injection. The precautions taken and the methods used for diagnosis and for blood and urine analysis were the same as recorded previously.[2]

RESULTS

The cows were grouped according to treatment and again subdivided into those which exhibited good recovery from one treatment and those which required retreatment (table 1). The average blood glucose and urine ketone qualitative (range 0-4) are shown on the day of treatment, samples being drawn just before treatment, and on the days indicated post-treatment. Most of the cows exhibited a good to excellent clinical response within 24 hours following treatment. However, approximately half of the cows which received 0.5 to 1.0 Gm. of hydrocortisone were retreated from the third to the tenth day, due to a failure to show continual improvement. Supplementation of the hydrocortisone treatment with glucose and calcium did not appear to have any advantage over that of hydrocortisone alone. For example, when single injections of hydrocortisone of 0.5 to 1.5 Gm. were given, 22 of the 36 cows not receiving calcium and glucose recovered without further treatment, whereas only 8 of the 25 cows which received the calcium and glucose supplement recovered without

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TABLE 1—Response of Cows with Ketosis in Los Angeles Area to Hydrocortisone Therapy

No. of cases	Clinical response		Blood glucose (G) in mg./100 ml. and urine ketone qualitative test (K) on days post-treatment.* Number of samples in parentheses				
			0	1	2-3	4-6	7-10
			Treatment: 0.5 Gm. hydrocortisone				
5	Good recovery	G	45.5(3)	57.0(2)	.....	49.5(3)	49.5(2)
		K	3.0(3)	2.0(2)	.....	1.3(3)	1.0(2)
3	Re-treatment	G	30.7(3)	60.7(3)	45.6(2)	.....	.....
	required	K	4.0(3)	4.0(3)	3.5(2)	.....	.....
			Treatment: 0.5 Gm. hydrocortisone plus 150.0 Gm. glucose with calcium				
4	Good recovery	G	35.0(4)	72.0(3)	49.3(3)	51.0(2)	56.7(3)
		K	3.8(4)	2.7(3)	2.0(3)	2.0(2)	1.0(3)
7	Re-treatment	G	34.4(7)	60.6(4)	44.0(5)	41.0(3)	29.9(4)
	required	K	4.0(7)	3.5(4)	2.2(5)	3.7(3)	3.5(4)
			Treatment: 1.0 Gm. hydrocortisone				
5	Good recovery	G	40.3(3)	65.5(3)	.....	51.0(3)	.....
		K	3.7(3)	2.5(3)	.....	1.0(3)	.....
3	Re-treatment	G	29.7(3)	61.0(2)	.....	38.7(3)	.....
	required	K	3.7(3)	3.0(2)	.....	2.7(3)	.....
			Treatment: 1.5 Gm. hydrocortisone				
4	Good recovery	G	38.3(4)	71.0(3)	54.3(3)	49.3(3)	.....
		K	4.0(4)	3.7(3)	0.7(3)	0.0(3)	.....
2	Re-treatment	G	37.5(2)	70.0(2)	.....	41.5(2)	.....
	required	K	4.0(2)	3.5(2)	.....	4.0(2)	.....
			Treatment: 0.25 Gm. hydrocortisone plus 0.25 Gm. hydrocortisone acetate				
5	Good recovery	G	33.0(3)	52.3(3)	.....	40.3(3)	.....
		K	3.7(3)	3.3(3)	.....	1.7(3)	.....
3	Re-treatment	G	31.3(3)	51.0(3)	.....	40.0(3)	26.5(2)
	required	K	3.7(3)	3.7(3)	.....	2.7(3)	3.5(2)
			Treatment: 0.25 Gm. hydrocortisone plus 0.25 Gm. hydrocortisone acetate plus 150.0 Gm. glucose with calcium				
2	Good recovery	G	32.5(2)	49.5(2)	.....	43.5(2)	.....
		K	4.0(2)	4.0(2)	.....	2.0(2)	.....
6	Re-treatment	G	24.7(6)	46.0(2)	50.2(4)	25.8(5)	24.0(2)
	required	K	4.0(6)	4.0(2)	4.0(4)	3.6(4)	4.0(2)
			Treatment: 0.5 Gm. hydrocortisone plus 0.5 Gm. hydrocortisone acetate				
9	Good recovery	G	37.3(9)	55.2(6)	61.0(4)	48.4(7)	.....
		K	4.0(9)	3.0(6)	1.0(4)	0.7(7)	.....
3	Re-treatment	G	30.5(3)	53.0(2)	28.5(2)	34.4(3)	.....
	required	K	4.0(3)	3.5(2)	3.0(2)	3.5(3)	.....
			Treatment: 0.5 Gm. hydrocortisone plus 0.5 Gm. hydrocortisone acetate plus 150.0 Gm. glucose with calcium				
2	Good recovery	G	29.0(2)	.....	.....	38.5(2)	.....
		K	3.5(2)	.....	.....	1.0(2)	.....
4	Re-treatment	G	28.0(4)	48.5(2)	37.7(3)	.....	25.2(4)
	required	K	4.0(4)	3.5(2)	3.3(3)	.....	2.5(4)
			Treatment: hydrocortisone plus hydrocortisone acetate (1:1), 1.0 Gm. on day 0 and 0.5 Gm. on day 1				
5	Good recovery	G	35.4(5)	69.3(3)	57.3(3)	47.5(3)	.....
		K	3.8(5)	2.7(3)	0.3(3)	1.0(3)	.....
1	Re-treatment	G	25.5	Good initial response		.....	18.8
	required	K	4.0			.....	4.0 (17th day)
			Treatment: hydrocortisone plus hydrocortisone acetate (1:1), 1.0 Gm. on day 0 plus 0.5 Gm. on days 1 and 2				
7	Good recovery	G	34.8(7)	.....	54.0(5)	69.6(3)	50.0(4)
		K	3.7(7)	.....	1.6(5)	0.7(3)	0.3(4)
			Treatment: hydrocortisone plus hydrocortisone acetate (1:1), 1.5 Gm. on day 0 plus 1.0 Gm. on day 1				
5	Good recovery	G	22.6(5)	59.0(3)	.....	49.0(5)	.....
		K	4.0(5)	3.0(3)	.....	0.4(5)	.....

*Data following re-treatment not included in table.

further treatment. The combination of hydrocortisone and hydrocortisone acetate (1:1) did not appear to be superior to that of hydrocortisone alone, 16 of the 32 cows treated with the combination, with and without glucose and calcium supplementation, recovering without additional treatment as compared with 14 out of 30 which received hydrocortisone alone. Cows exhibiting both ketosis and parturient paresis were encountered but data on these animals are not included in this report.

Remarkable recovery was obtained, however, when an initial injection of 1.0 Gm. of the hydrocortisone combination was followed by 0.5 Gm. for the next two days and when an initial injection of 1.5 Gm. was followed by 1.0 Gm. on the following day,

the 12 cows so treated showing rapid recovery, with both blood glucose and urine ketones being in the normal range from the fourth to sixth days. The clinical response was especially good, the cows receiving 1.5 Gm. followed by 1.0 Gm. exhibiting the most rapid clinical recovery. Treatment with 1.0 Gm. followed by 0.5 Gm. also gave good results, 5 of 6 recovering without additional treatment; but the recovery of the 12 cows receiving the higher dosage was more rapid in most cases.

The blood ketones of the cows which exhibited good recovery (table 1) following the initial treatment were appreciably lower between the second and sixth days than in the case of the cows which required further treatment. Likewise, blood glucose was usually higher during this period in the cows which did not require additional treatment. At the higher dosages given for two or three days, the blood glucose was maintained at the relatively high level and the blood ketones cleared up quickly.

In view of the fact that the ketotic cows in the Los Angeles area required appreciably more hydrocortisone than those which were studied in the Maryland area, some observations were also made with ACTH and cortisone (table 2). Of 6 cows which received 600 A.V.U. of ACTH, 3 recovered and 3 required additional treatment. Of 8 cows which received an initial injection of 400 A.V.U. of ACTH, followed by 200 on the following day, 4 recovered and 4 required additional treatment, thus showing that more ACTH was also needed for the successful treatment of ketotic cows in the Los Angeles area than for most cases in the Maryland area.

To obtain additional information on the efficacy of relatively large doses of ACTH and glucocorticoids, 2 cows were studied which had a history of being refractory in previous years. They were in the same herd and each had received 5 treatments with 500 ml. of 50 per cent glucose with but slight response, the last injection of glucose in each case being made five days previous to the use of ACTH and cortisone. The regimen for 1 cow was an initial injection of 600 A.V.U. of ACTH on day 0, 300 on day 1, and 300 on day 3, and for the second cow 1.5 Gm. of cortisone acetate on day 0 followed by 1.0 Gm. on day 1 and 1.5 Gm. on day 3. Both cows recovered rapidly, appearing completely normal by the fourth day. The urine ketones were negative in both animals on the fourth day and remained negative thereafter. The results are believed to be significant in showing that even the refractory cases will respond rapidly, in most cases, to glucocorticoids or ACTH.

Two cows proved particularly refractive to hydrocortisone and ACTH (table 3). Cow A received hydrocortisone, glucose, and ACTH over a period of two weeks, but maintained a relatively low level of blood sugar and high level of blood ketones throughout the entire period. The appetite did improve during this period, but milk production remained low. It appears likely that the same amount of hydrocortisone

TABLE 2—Response of Cows with Ketosis in Los Angeles Area to ACTH and Cortisone Therapy

No. of cases	Clinical response		Blood glucose (G) in mg./100 ml. and urine ketone qualitative test (K) on days post-treatment. Number of samples in parentheses			
			0	1	2-3	4-6
Treatment: 600 A.V.U. ACTH*						
3	Good recovery	G	37.1(3)	.....	.....	45.8(3)
		K	4.0(3)	.....	.....	0.5(3)
3	Re-treatment required	G	24.9(3)	41.8(3)	.....	27.4(3)
		K	4.0(3)	3.7(3)	.....	4.0(3)
Treatment: 400 A.V.U. ACTH on day 0 plus 200 A.V.U. on day 1						
4	Good recovery	G	33.2(4)	.....	80.1(3)	46.1(4)
		K	3.7(4)	.....	3.0(3)	1.0(4)
4	Re-treatment required	G	33.2(4)	.....	38.8(3)	25.7(4)
		K	4.0(4)	.....	2.7(3)	3.5(4)
Treatment: A.V.U. ACTH, 600 on day 0 plus 300 on day 1 plus 300 on day 3 Previously received 5 treatments with 500 ml. 50% glucose						
1	Good recovery	G	28.6	.....	44.6	46.8
		K	4.0	.....	3.0	0.0
Treatment: cortisone acetate, 1.5 Gm. on day 0 plus 1.0 Gm. on day 1 plus 1.5 Gm. on day 3 Previously received 5 treatments with 500 ml. 50% glucose						
1	Good recovery	G	35.4	.....	41.2	48.7
		K	4.0	.....	3.0	0.0

*Armour adrenomone.

administered over a period of two to three days would have been far more effective. Cow B received ACTH over a period of 11 days without exhibiting recovery, the blood glucose being relatively low during most of the period and the urine ketones high. The

TABLE 3—Blood Glucose (G) in mg./100 ml. and Urine Ketone Qualitative Test (K) of Cows Resistant to Treatment

Cow A			Cow B		
Days	G	K	Days	G	K
0-0.5 Gm. F* + 300 ml. 50% glucose	19.4	4	0-400 A.V.U. ACTH**	23.5	4
6-0.5 Gm. F + 500 ml. 50% glucose	17.0	4	1-200 A.V.U. ACTH	.....	..
7-0.5 Gm. F	.....	..	2-	25.3	4
8-0.5 Gm. F	.....	..	4-600 A.V.U. ACTH	48.5	4
12-400 A.V.U. ACTH	30.7	4	5-200 A.V.U. ACTH	.....	..
13-200 A.V.U. ACTH	.....	..	7-200 A.V.U. ACTH	.....	..
15-	25.3	3	9-500 A.V.U. ACTH	35.6	4
			11-300 A.V.U. ACTH	.....	..
			15-	54.2	4

*Hydrocortisone plus hydrocortisone acetate 1:1. **Armour adrenomone.

appetite of this cow did improve appreciably but milk production remained low. In this cow, at least, it appears that the adrenal gland may have been relatively unresponsive.

Most of the cows which did not recover following a single injection of hydrocortisone or ACTH did recover following one to two later treatments, but the response was not as rapid as when a similar or lesser total dosage was administered at one time or over a period of two to four days. Many animals which exhibited a good clinical response followed by a relapse, and which were not re-treated until the condition was again rather acute, had to be treated like new cases and often required more hydrocortisone than was used in the initial treatment to obtain a similar response. On the other hand, cows which exhibited a good initial response and which later showed rather mild signs of clinical ketosis usually recovered rapidly following the injection of 0.5 Gm. of hydrocortisone.

DISCUSSION

The results show that cows with ketosis in the Los Angeles area, on the average, require appreciably more glucocorticoids and ACTH than cows which have been studied in the Maryland area. It appears entirely possible that there may be other areas, or specific herds, or individual animals in other areas in which this same situation exists. It is apparent that even most of the refractory cases will exhibit good recovery when relatively large doses of glucocorticoids or ACTH are used. This should become particularly important when some of the present glucocorticoids are available at a lower price or when some of the new, highly active glucocorticoids become available. It is especially interesting that the most rapid and permanent recoveries following the use of glucocorticoids and ACTH are accompanied by a rapid decrease in the urine ketones. Urine ketones which remain high four days or more following treatment with glucocorticoids or ACTH appeared to be indicative of animals which may be expected to exhibit clinical relapse or slow recovery. In fact, it appears that glucose values beyond the first day may not be necessary for the proper evaluation of either glucocorticoids or ACTH.

Records which were obtained on the origin of 79 of the ketotic cows observed in this study showed that 57 per cent had been shipped into the area immediately prepartum, whereas Los Angeles County records show that only one-third of the cows in this area were replaced each year during the two years preceding this study. It is evident, therefore, that the incidence of ketosis was appreciably higher in the cows which were shipped just prior to parturition. The percentage of cows refractory to treatment was almost equally divided between the local cows and the cows shipped into the area. No relationship was observed between feeding regimens and the incidence of ketosis.

Practically all of the cows received an abundance of excellent quality alfalfa hay or green-cut alfalfa or both and an abundance of good concentrates, most of the milking cows receiving 14 to 16 lb. of concentrate daily beginning almost immediately postpartum. During the dry period, most of the native cows received an excellent quality alfalfa hay with or without molasses *ad libitum* and some received fresh-cut alfalfa or were on pasture. The cows were primarily high-producing Holstein-Friesians, the majority having had 1 or more calves. The stress of shipping and change in environment may have been an important factor in the development of

ketosis in the cows which were shipped into the area just prior to parturition. Likewise, a change in the feeding regimen would naturally occur. It is also possible that the stress of high production and heavy feeding may have been an important factor in the development of ketosis in the cows which had been in the herd for one or more years. The incidence of ketosis in a number of nearby herds on excellent irrigated pastures appeared to be about as high as that of the cows fed in drylots. Likewise, the feeding of large amounts of freshly cut alfalfa did not diminish the incidence of ketosis.

CONCLUSIONS

Hydrocortisone and a combination of hydrocortisone and hydrocortisone acetate proved to be highly effective for the treatment of bovine ketosis in the Los Angeles area when used in adequate amounts. Almost twice as much glucocorticoids and ACTH were required to obtain consistent results as was found to be adequate in the Maryland area. Rapid recoveries were obtained with refractory cases by the injection of ample amounts of glucocorticoids or ACTH over two- to four-day periods. The incidence of ketosis was highest in the cows shipped into the area immediately prepartum.

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Pasteurella in Shipping Fever

In a further report by Carter and McSherry (*Canad. J. Comp. Med.*, June, 1955) on 33 young cattle with shipping fever, *Pasteurella hemolytica* was recovered from the nasal passages of 27 and pleuropneumonia-like organisms from 16 of the 33. No characteristic changes in the white cells were observed. As no organisms were found in the blood of 7 other animals, it was not a septicemia.

Ketosis Confusion Clearing

R. C. KLUSSENDORF, D.V.M.

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Ketosis, or acetonemia, has long been a perplexing problem. Perhaps recognizing the complex nature of the condition is an important step toward solving it. Among the things we have learned about its treatment and prevention are:

1) Glucose solution injected into the vein is the most common treatment. It is a dependable way to relieve the patient, but it wears off quickly. It is not used preventively.[1]

2) Cortisone and the corticotrophic hormones have produced a favorable response. These cost more than glucose but their effects last longer. Again, they have not been used preventively.[2]

3) Propionate and acetate of sodium may be fed to cows with mild ketosis with beneficial results. They are just being tried as preventives.[3]

4) Preventive steps have been more erratic than treatments but could become highly effective if new warning tests are used widely.[4]

A NEW STEP

A fresh angle is suggested by Dr. E. S. Gordon, of the College of Medicine at the University of Wisconsin, who believes that ketosis is a manifestation of a stress phenomenon.[5]

What is a stress phenomenon? Fundamentally, it is a general pattern of behavior shown by all warm-blooded animals when they are exposed for a long time to any unusual and exhausting condition.

What causes stress? Stress may be provoked in many ways, among which are: trauma (accidental, surgical, obstetrical), violent muscular exercise, infection (bacterial, viral), hemorrhage, cold, fever, anoxia, burns, a poison or drug or hormone in abnormally large doses, radiation, or nervous shock.

How does stress develop? It develops in three stages to produce a pattern which the doctor calls a "syndrome." The first stage is an alarm reaction which appears in two phases (shock and counter shock); next comes the stage of resistance and, finally, the stage of exhaustion.

During the alarm reaction, structural and functional changes occur in many organs and tissues, so that certain metabolic disturbances result. The basic change rec-

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ognized in ketosis is failure or inability to metabolize fats normally or completely, leaving a residue of *beta* hydroxybutyric acid. Such a pattern is also shown when ACTH and cortisone are in short supply. Whether endocrine deficiency is really present in ketosis remains to be proved, but the prompt response to injection of the adrenal cortical hormones lends plausibility to the hypothesis of stress and endocrine imbalance being fundamental in ketosis as an expression of the alarm reaction.

During the stage of resistance, the symptoms subside or disappear. Many ketosis cases recover spontaneously, the system resisting to the point of recovery without treatment.

The stage of exhaustion leads to reappearance of the symptoms, often in a more severe form.

When viewed in the light of the stress reactions, ketosis appears to be a metabolic anomaly induced by causes which no one understands and which may have nothing to do with subnormal adrenal function, and perhaps not with inactivity of the pituitary gland. Let's see what happens.

At parturition, the dairy cow suffers from hypoglycemia—reduced blood sugar. This may vary from a mild case, in which blood sugar is lowered only 20 to 30 per cent, to a severe one, in which blood sugar may be down to 50 per cent, or less, of normal.

For some reason, the normal body metabolism is maintained during a period of stress only if somewhat increased amounts of adrenal cortical hormones are present. On the other hand, overactivity of the adrenal glands or injection of an excess of adrenal cortical hormones causes a disturbed metabolism of water, salt, carbohydrate and protein, and results in resistance to many physical and chemical agents and infections. The latter probably is the important factor in ketosis. In other words, the system is placed in a state of reduced sensibility, so that the symptoms are not shown, even though the basic metabolic imbalance has not been corrected. We know that many cows with ketosis will live and recover completely without treatment, so the extra adrenal cortical hormone may keep the cow on an even keel while nature corrects the basic imbalance.

Stress may be accompanied by increased sugar production, increased nitrogen output in urine, and many other phenomena.

Fat metabolism is altered when the adrenal gland is stimulated. The degradation of ketone bodies continues to its normal termination, or the precursors of the ketone bodies may be metabolized without going through the *beta* hydroxybutyric acid stage.

Other endocrine glands and secretions are affected by the stress reaction, and the pituitary gland changes may be involved in ketosis, since it occurs chiefly in pregnant and recently pregnant animals in which profound changes in anterior pituitary functions have already occurred.

PREVENTION

The presence of excessive quantities of *beta* hydroxybutyric acid in the blood has been mentioned by physiologists as the cause of many of the symptoms of ketosis. Hence, there is logic for looking again at the metabolic balance in the rumen—and specifically the production of added amounts of propionic acid to balance or neutralize, or to prevent the production of excessive amounts of *beta* hydroxybutyric acid.

The basis for administering sodium propionate seems to lie in this direction, and suggests that prevention might be more effective than treatment. Recent work[6] suggests three possible uses of propionate—as treatment, to prevent relapse, and as preventive. A study of the tables and curves which accompany this report[6] suggests that a lag period exists between administration of the first dose and noticeable improvement in decreasing blood ketones, increasing blood sugar, and producing more milk.

The logical approach, if this is true, would be to feed small amounts of the propionate in the fitting ration during the four or six weeks before calving, to condition the rumen and its microorganisms and enable them to make prompt and full use of therapeutic amounts should they be needed.

Since sodium propionate is distasteful to cows, various levels will need to be tried in the feed, or ways to mask the taste may be found. Also, work at Iowa State College[7] has shown that a related chemical, propionamide, is not distasteful, and this may be a better product for preventive use.

The net result of the review, then, is a pair of questions: (1) Is the stress reac-

tion an important factor in ketosis? (2) Is the maintenance of normal rumen metabolism a step toward preventing or reducing the stress of pregnancy and parturition? The answers are not now avaliable.

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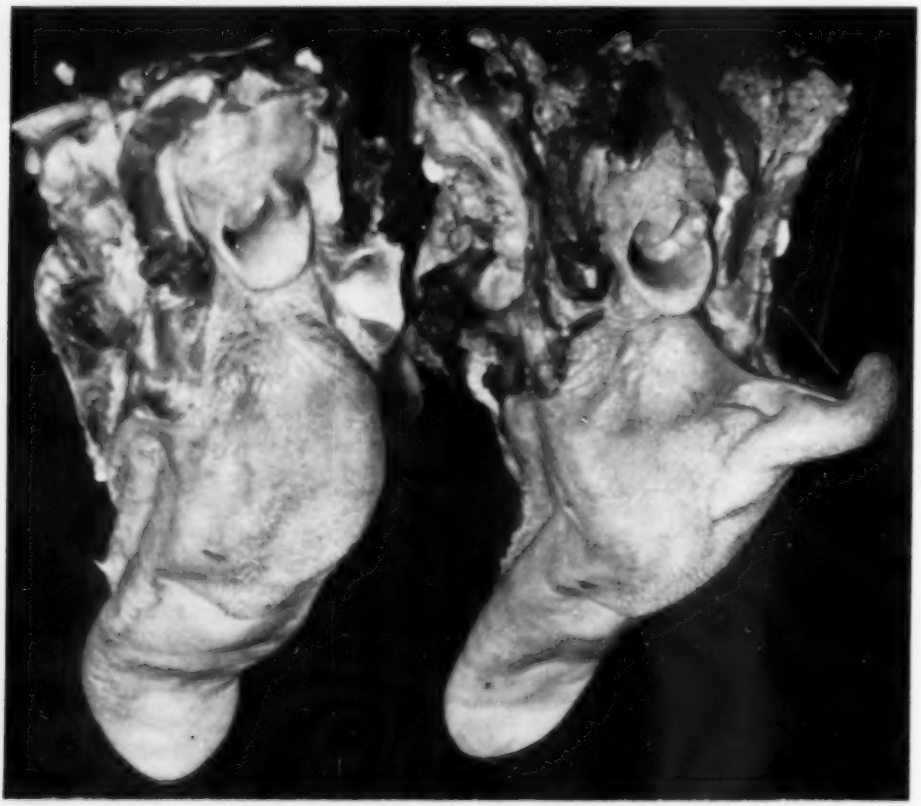
Horse Serum Shock in Bulls.—When 2 yearling bulls (half brothers), with no known previous serum treatment, were each given 6 ml. of tetanus serum, both developed serum shock and 1 died.—*Vet. Bull., May, 1955.*



Supernumerary tongue (right) from an Aberdeen Angus heifer which had given no evidence of coughing, choking, or other ill-effects, shown with a normal bovine tongue (left). The rudimentary second tongue bore filiform and fungiform papillae, and the bulge at the root of the main tongue caused the food to pass through the right side of the pharynx. The photograph was submitted by Dr. Walter Wirzsczuk, U.S.D.A. inspector at the Elburn Packing Company, Elburn, Ill.

Summaries of Experiments in Swine Erysipelas in Germany

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DURING RECENT YEARS experimental work on various phases of the epidemiology and pathogenesis of swine erysipelas has been conducted at the Veterinary Department of the Bundesgesundheitsamt at Berlin. The results of these investigations are condensed and compiled in this article.

ERYSIPELAS ORGANISMS IN THE SKIN AND BLOOD

The blood of swine, experimentally infected by the percutaneous (scarification) method of Fortner and Dinter, was examined daily, culturally and by injection into white mice, starting the day of the infection. In some cases, the blood was examined every two hours during the first 24 hours. In animals with only a local reaction along the scarified area, the erysipelas organism could not be detected in the blood, even after several days of high temperature.

In the majority of animals which showed diamond skin lesions (urticarial wheals) in addition to the local reaction, erysipelas organisms could be demonstrated in the blood for one to several days. Those in which no bacteremia could be detected had only a few diamond skin lesions. It may be assumed that in these pigs the bacteremia either was so slight or it existed for such a short time that it eluded detection. (Possibly, a small number of the bacteria might be found in the blood of animals without causing the formation of diamond skin lesions.) When animals died in about two or three days with a fatal septicemic infection, there was almost always an increasing number of bacteria in the blood stream. However, in the case of prolonged sickness, the bacteremia might greatly diminish.

Histologically, the local reaction in the skin manifests a great invasion of bacteria in the tissue of the papillary body, in the corium, and into the subcutis. A similar invasion occurs in the diamond skin lesions. The bacteria are also detectable culturally or by injection of skin scrapings into mice or swine. (In neither the local reaction nor the urticarial swelling could bacteria be found in the local blood capillaries. Conversely, in the erythematous skin, many bacteria are found in the capillaries with fewer in the tissue.) In scarified areas which heal without reaction, a slight infection can be recognized.

CHRONIC ERYSIPELAS

Experimental studies on chronic erysipelas made it evident that arthritis and endocarditis occur only after a bacteremia, *i.e.*, only in pigs which have developed urticaria or which have received an intravenous injection of living erysipelas bacteria. The repeated intravenous injection of dead erysipelas organisms into swine which had never been affected with generalized erysipelas did not produce these chronic erysipelas lesions. This suggests that the lesions of chronic erysipelas (arthritis or endocarditis) are the results of erysipelas bacteria multiplying in these tissues. In rats, chronic erysipelas could be produced by one simple infection.

In swine suffering from erysipelas arthritis, the bacteria are frequently found in the corresponding lymph nodes. In animals with endocarditis, they are found in the blood. The erysipelas bacteria found in the chronically diseased organs are only slightly pathogenic for mice and swine, but when they are cultivated in the organ material of the killed animal or in an artificial culture medium, they can develop an increased pathogenicity.

Some authors are of the opinion that allergies are mainly responsible for producing acute and chronic erysipelas. However, no indication of an allergy was found in the experimental erysipelas tests made on more than 1,000 pigs.

HEREDITARY SUSCEPTIBILITY

Within the litters of the swine in this Institute (Veterinary Department of the Bundesgesundheitsamt, Berlin), the pathological signs occurring after percutaneous inoculation were strikingly uniform. The

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This paper was translated from the German by Gertrude D. Schwerin, AVMA librarian.

Dr. Wellmann, who recently studied in this country on a Fulbright Fellowship, is professor and head of a research laboratory for veterinary zoology and bacteriology of the Federal Health Office, Max von Pettenkofer Institute in Berlin, Germany.

only distinct differences were between the families, according to their hereditary resistance. Within the litters which had been bought from outside of the Institute, the signs of infection varied from none to septicemic erysipelas. Some entire litters were refractory, while their offspring were susceptible. Similar phenomena were observed after spontaneous erysipelas had occurred in the different families of the Institute swine. This phenomena could be explained only by specific antibodies having originated from a subclinical or an abortive infection.

The author developed serological research methods which made it possible to recognize the infection which passed through the body and to prove the existence of antibodies. One method is the growth test. Sterile, native blood serum of the animal to be tested is diluted—at various levels—with horse meat infusion broth. Into this broth is injected an erysipelas strain which renders a stock culture (a 10% horse serum broth) uniformly turbid without forming a sediment. Serums of swine which prove to be highly susceptible to the percutaneous infection are uniformly turbid even in low level dilutions, while serums of refractory (immune) animals show a growth by the formation of a flocculent sediment. This method is effectively completed by the hemagglutination-inhibition test.

The author was also able to produce subclinical infections, which might make the animal immune, by means of slightly pathogenic strains used percutaneously and alimentarily, and by means of highly pathogenic strains rubbed into the scarified mucosa of the upper lip. Transmission could be also affected through houseflies.

A further possibility of transferring immune bodies to sucking pigs is through the colostrum of immunized sows.

As frequently proved before, the erysipelas bacterium is ubiquitous. It has been isolated from the tonsils of swine from herds in which no erysipelas has been observed for many years and also from saltwater fish. These strains usually had only a slight pathogenicity for swine, yet in nature they are probably able to immunize and to induce subclinical or abortive infections.

The author concludes from the results of these investigations that there are three main factors which influence the development and the course of erysipelas infection in a pig: (1) hereditary susceptibility; (2) the infection modus; and (3) the pathogenic quality of the erysipelas bacteria.

INTERMEDIATE CARRIERS OF ERYSIPELAS

To determine whether other animal species can be intermediate carriers (vectors) or reservoirs, the susceptibility to erysipelas of 15 species of Muridae (rodents) was investigated. Some species were highly susceptible, others were more or less resistant. Among the more resistant species were those which did not visibly contract the disease but which showed bacteremia; such rodents can be carriers and eliminators of the bacteria. The sparrow, likewise, proved to be markedly susceptible. It could be easily infected alimentarily.

Insects also can be vectors. It was relatively easy to transmit erysipelas infections to laboratory animals and to swine through stinging insects as well as through the nonstinging housefly. The source of infection for the insects was infected animals with bacteremia or material infected artificially with bacterial culture. The infection produced in swine by insect transmission is at times subclinical and can be proved only by an immunity test.

There is no proof that insects are highly significant in the transmission of erysipelas, nor that rodents and wild birds are important as intermediate carriers or epizootic reservoirs. Yet the possibility should be considered at all times.

LONGEVITY OF ERYSIPELOTHRIX RHUSIOPATHIAE

The remarkable life span of the erysipelas bacterium is shown by the fact that a 22-year-old erysipelas broth culture, not even conserved at low temperatures, but exposed to the temperature variations of the seasons, was still able to kill mice and to produce in swine, percutaneously, a local skin reaction leading to immunization.

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Odd Viruses in Swine Influenza.—In the first of two series of studies of swine influenza in Poland, the eight strains of virus isolated were identified as Newcastle disease virus. In the second series a virus designated "G_1", resembling Shope's virus, was isolated.—*Vet. Bull., July, 1955.*

Effect of DDT-Treated Feed on Animals

When one third of the feed given pigs was alfalfa with 23 p.p.m. of DDT residue, they remained normal. When their fat and flesh, which contained 2.2 to 5.3 of DDT, was fed to young rats for 14 weeks, they also remained normal.—*Vet. Bull., June, 1955.*

Chicks Failed to Respond to Antibiotics

Penicillin or aureomycin® failed to increase the growth rate of chicks after June, 1952, whereas previously, in the old environment, they had been effective. This could have been due to a change in sanitary and cleaning practices, or harmful bacteria may have been eliminated by the long use of antibiotics.—*Vet. Bull., June, 1955.*

Another Idea on Photosensitization

That aphids (plant lice) might be associated with the production of photosensitization of animals has been suggested by nutritionists in the *Australian Veterinary Journal* (April, 1955).

The prevalence of aphids in a part of New South Wales, where this allergic condition is common in herbivores, has caused the name "aphis disease" to persist, although scientists indicated nearly 40 years ago that the plants involved could alone be responsible.

It is now reported that some aphids contain the fluorescent pigment, erythroaphin, which can promote photodynamic hemolysis and which is similar to some of the photosenaitizing plant pigments.

Whether aphids have ever been similarly suspected on this continent we do not know, but before dismissing the thought a few quick "looks" might be wise. It should be remembered that farmers who fed white corn to cattle believed it inferior to yellow corn for many years before the former was found to be inadequate in vitamin A content.—W.A.A.

Milk in a clear glass container in a sunny place loses riboflavin and may develop an off-flavor in half an hour.—*Sci. News Letter, July 16, 1955.*

Symposium on Granulomatous Diseases—Part I

AT THEIR annual seminar, members of the American College of Veterinary Pathologists study the pathological changes in specimens from selected cases. After studying the sections and case histories, each member makes his diagnosis.

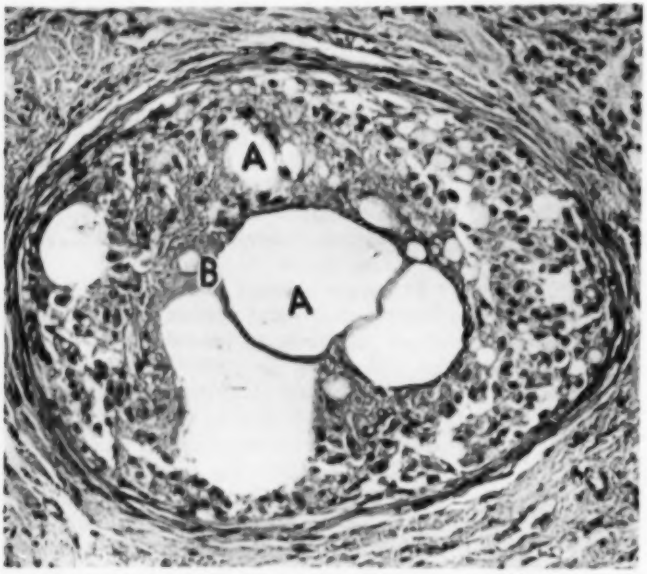
Since these are problems in clinical diagnosis, many of which should be of interest to all veterinarians, the College has submitted briefed case histories, illustrations, comments, and diagnoses for publication.

The subject discussed at the fifth annual seminar of the College, held in Chicago, Nov. 27, 1954, was granulomatous diseases. Summaries of some of the cases presented will appear in the JOURNAL periodically.

CASE 1—FOREIGN BODY GRANULOMA DUE TO MINERAL OIL—*G. R. Spencer, D.V.M., Ph.D.*

History.—A 5-year-old Labrador Retriever bitch was treated for entropion of the lower lids by injecting mineral oil. Four months later, hard swellings appeared at the sites of injection. These were removed surgically but recurred in about a month. The growths were removed four times and specimens of the fourth removal were submitted for histological diagnosis.* The nodules were 2.5 cm. in diameter and aggregates of white, tough tissue were found.

Comments (Moderator, Lt. Col. T. C. Jones).—The whole section included fragments of skeletal muscle which were infiltrated and in some instances displaced by collections of histiocytic cells. In some places, these histiocytic cells formed circumscribed nodules (fig. 1), a striking feature of which, even at low power, was the presence of many smoothly spherical spaces (fig. 1 [a]) of various sizes scattered through the histiocytic tissue (probably oil spaces). At a few points adjacent to the large vacuoles there was some hyalin material (fig. 1 [b]). Sections specially stained for fat (oil red O) and acid-fast organisms were negative for both.



—A.F.I.P.—54-21388

Fig. 1—This foreign body granuloma (case 1), removed from the lower eyelid of a Labrador Retriever was caused by injected mineral oil. Notice the connective tissue encircling the nodule. The nodule is composed of fibrous tissue some of which is hyalinized (b), histiocytes, and spherical clear spaces (a). The clear spaces probably once contained oil droplets. x 220.

Diagnosis.—The diagnosis was lipogranuloma due to injection of mineral oil to correct an entropion.

Case 1 was presented by Dr. G. R. Spencer of the College of Veterinary Medicine, State College of Washington, Pullman.

*The tissue was received from Dr. A. J. Ryncarz, Tacoma, Wash.

CASE 2—FIBROPAPILLOMA OF THE PENIS OF A BULL—*R. M. Roderick, D.V.M., Ph.D.*

History.—A single localized granulomatous lesion,§ 6 by 6 by 4 cm., was attached to the lateral surface of the glans penis of a bull by a projecting stalk of tissue. The lesion was cauliflower-like and raised from the surface of the organ (fig. 2).

Comments (Moderator Jones).—The section enclosed a mass which was ulcerated in places. Most of the surface (fig. 3) was covered by a thick stratified squamous epithelium which in some places was cornified with retention of nuclei (parakeratosis). The epithelium (fig. 3 [a]) sent long branching fronds deep into the underlying stroma. The bulk of the mass was made up of interlacing bundles of spindle-shaped cells (fig. 3 [b]) which in some places were admixed with leukocytes. In the deeper parts of the lesion, the spindle-shaped cells formed differentiated fibrous connective tissue. In some of the deeper areas, the cytoplasm of the cells contained strands of collagen, but in other places there was little or none. There were no collections of histiocytic cells, nor any abscesses or leukocytes other than those seen near the ulcerated surface.

Case 2 was presented by Dr. R. M. Roderick of the School of Veterinary Medicine, Kansas State College, Manhattan.

§The tissue was received from Dr. E. R. Frank, Department of Clinics, Kansas State College, Manhattan.

Diagnosis.—The diagnosis was transmissible fibropapilloma of the bovine genitalia.

Fig. 2—Transmissible fibropapilloma of the bovine penis (case 2).

Discussion (Moderator Jones).—This was probably not malignant. The lesion had some histological and clinical similarity to "equine sarcoid."

Dr. Kenneth McEntee (N.Y.).—This lesion was transmissible from animal to animal—from the male penis to the female vagina and vice versa. The lesion was caused by the bovine wart virus and, as it is self-limiting, it would eventually disappear. Treatment with wart vaccine has been used but without much success.

Dr. J. H. Sautter (Minn.).—These lesions may recur after surgery, often within two weeks. The recurrent lesions have a microscopic appearance suggesting fibrosarcoma.

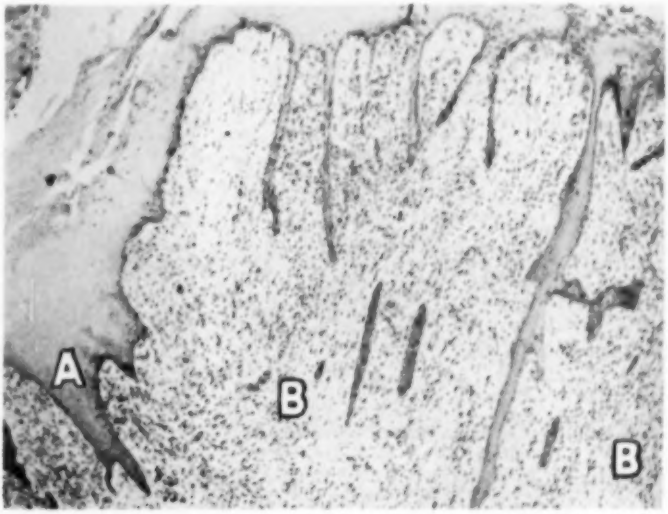


Fig. 3—A cross section of the lesion of the bovine penis (case 2). Notice the interlacing bundles of spindle-shaped connective tissue cells (b) which in some places are admixed with leukocytes. The papillary tufts are covered by stratified squamous epithelium (a) which has ulcerated in some places. x 67.

—A.F.I.P.—14-21806

Dr. L. M. Roderick (Kan.).—Although this lesion may recur, it is not malignant and does not metastasize.

CASE 3—ACTINOBACILLOSIS IN A COW—*Cecil Elder, D.V.M., M.S.*

History.—A 3-year-old Jersey which had a rumenotomy in August, 1953, was brought to the hospital in February, 1954, to have excessive granulation of the rumenotomy scar excised. There was no recurrence of the growth.

Comments (Moderator Jones).—This section included a fragment of collagen-rich connective tissue in which there were several microabscesses which in turn were surrounded by histiocytes. In the center of these abscesses were colonies of organisms which were brightly eosinophilic and which had a radiating clublike structure (fig. 4). These bodies were believed to be colonies of organisms. The internal structure in the sections stained with hematoxylin and eosin revealed indistinctly stained, small bacillary forms. Gram-stained sections (Braun and Brenn) showed the interior of the colony to be filled with gram-negative organisms.

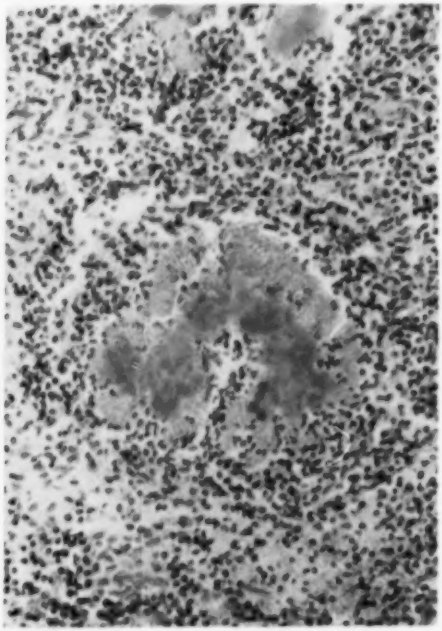
Diagnosis.—This condition was diagnosed as actinobacillosis at site of the abdominal surgical wound.

Discussion.—(Dr. C. L. Davis, Colo.): About 95 per cent of the lesions in soft tissues in cattle are actinobacillosis rather than actinomycosis. The gross appearance of lesions of actinobacillosis, particularly when they involve cervical lymph nodes, may suggest tuberculosis. Microscopic differentiation of the lesions, particularly when special stains are used to demonstrate the organisms, are necessary in making diagnoses.

CASE 4—COCCIDIOIDOMYCOSIS IN A DOG—*R. F. Langham, D.V.M., Ph.D.*

History.—This dog was purchased in New Mexico and brought to Michigan. After several months, the dog developed lameness and a swollen joint in a front leg. From the joint, a fungus was isolated.

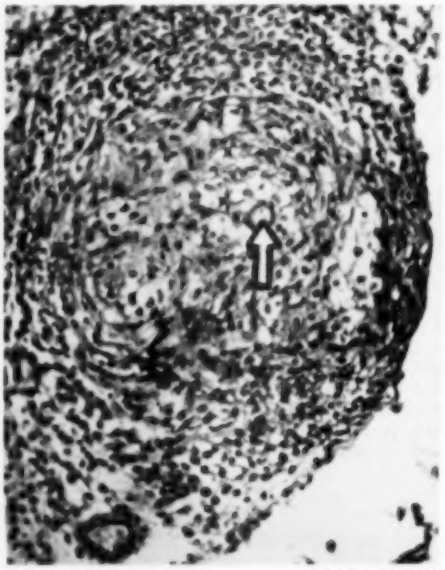
Case 3 was presented by Dr. Cecil Elder of the Department of Veterinary Science, University of Missouri, Columbia.



—*A.F.I.P.—54-21808*

Fig. 4—Colony of Actinobacillus organisms (Actinobacillus liqnieresi), brightly eosinophilic, surrounded by histiocytes (case 3). x 210.

Case 4 was presented by Dr. R. F. Langham of the Department of Animal Pathology, Michigan State University, East Lansing.



—*A.F.I.P.—54-21812*

Fig. 5—Section of lymph node (case 4) showing a granuloma and microabscess. In the center of the foci is a spherical coccidioidomycosis organism (arrow) which has a thick wall. x 210.

In spite of several week's treatment, the dog lost weight and died. At necropsy, nodular lesions were found in the lungs, brain, heart, liver, and some lymph nodes (mediastinal, bronchial and mesenteric). The lesions varied from 1 to 60 mm. in diameter, were yellowish gray, and many were calcified.

Comments (Moderator Jones).—In the lymph node, most of the lymphatic structures were displaced by small and large granulomas, some individually discrete and others confluent. There was a liberal admixture of neutrophils throughout the lesion and in some places microabscesses were formed. In many places, the histiocytic cells were densely packed and surrounded by a tiny focus of neutrophils (fig. 5). In the center of a few such foci were seen a spherical organism with a double-contoured wall (fig. 5). These measured approximately 25 μ in diameter, showed no evidence of budding, and some contained endospores.

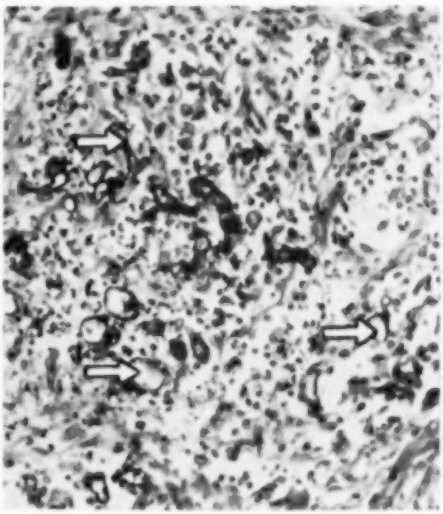
Diagnosis.—The diagnosis was generalized canine coccidioidomycosis.

Discussion (Dr. C. L. Davis, Colo).—I have seen lesions of two year's duration in the thoracic lymph nodes in a cow. They were calcified but it was still possible to culture *Coccidioides immitis* from them.

CASE 5—MUCORMYCOSIS AND DISTEMPER IN A DOG—*Lt. Col. Chester A. Gleiser, V.C.*

History.—A 3-month-old male Terrier showed signs of fever, diarrhea, anorexia, and pneumonia, followed by convulsions and death in 13 days. At necropsy, an ulcer 2 cm. in diameter was found in the fundus of the stomach. The intestines were thickened and the lungs were congested.

Comments (Moderator Jones).—This section was taken through the wall of the fundic portion of the stomach, probably near the pyloric region. The mucosa was eroded and replaced by a sharply delimited lesion which extended through the muscularis mucosa deep into the submucosa and in some areas actually invaded the muscularis. This lesion had necrotic debris on the surface, was richly infiltrated with neutrophils, and in deeper areas it had foci of necrosis plus large numbers of histiocytic cells in which there were numerous organisms. These organisms were large, extremely variable in size and shape, often showed budding, hyphae, and occasionally branching (fig. 6). The organisms had a thick, double-contoured wall, and were usually empty but occasionally contained granular material. The coarse, roughly spherical organisms measured 24 to 40 μ in diameter. The hyphae which extended from the large globular structures were usually about 8 μ in diameter. They branched but it was difficult to detect any septae. Occasional giant cells were seen engulfing organisms. The organisms were particulary well demonstrated in the sections when stained with the para-aminosalicylic reagent. They were also easily differentiated by Bauer's stain but Gridley fungus stain did not bring them out selectively. In sections stained with iron hematoxylin followed by van Gieson's stain, the organisms were particularly well demonstrated and differentiated from the background. Cytoplasmic inclusions, compatible with those of canine distemper, were also seen in the gastric epithelium.



—A.F.I.P.—54-21815

Fig. 6—Ulcer of stomach (case 5) due to Mucor sp., associated with canine distemper (stained by periodic acid Schiff reaction). Irregular-sized organisms of Mucor sp. (arrows) can be seen in granuloma adjacent to ulcer (irregular, round bodies in center of the photomicrograph). x 210.

Diagnosis.—The diagnosis was ulcer of

Case 5 was presented by Lt. Col. Chester A. Gleiser, V.C., U.S. Army, Washington, D.C. It was previously reported in J.A.V.M.A., 123, (1953): 441-445.

the stomach due to Mucor sp., probably associated with canine distemper.

Discussion (Dr. H. R. Seibold, Ala.).—There are eosinophilic cytoplasmic inclusions in the chief cells of the gastric glands at points distant from the mycotic lesion which suggest that canine distemper is involved in this case.

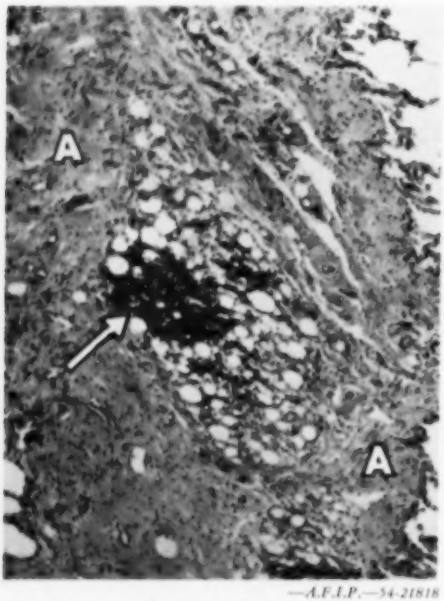
Dr. David L. Coffin (Mass.).—Distemper inclusions are found in the gastric epithelium, both in the cytoplasm and nuclei. I have been able to demonstrate fluorescent antibodies in connection with these inclusions.

Dr. J. H. Sautter (Minn.).—I seldom find inclusions in the stomach epithelium but I have found them in the epithelium of the intestine.

[Mucor infection is not considered very pathogenic but its growth is stimulated following the administration of antibiotics.—ED.]

CASE 6—FIBROSING CHRONIC ASPIRATION PNEUMONIA IN A COW—*W. A. Anderson, D.V.M., M.S.*

History.—A 9-year-old cow in poor physical condition was slaughtered in a federally inspected plant. The lungs and bronchial and mediastinal lymph nodes contained lesions resembling tuberculosis. No other lesions were observed.



—A.F.I.P.—54-21818

Fig. 7—Section of lung (case 6) showing areas of dense collagenous fibrosis pneumonia (A). Notice the circumscribed area showing a network of clear spaces (oil) and nests of lymphocytes (arrow). x 62.

Comments (Moderator Jones).—The section, from a prominently lobulated lung, showed large areas of dense collagenous connective tissue (fig. 7). This fibrosis appeared to invest some alveoli and alveolar ducts and to displace others. In many areas throughout this fibrous tissue, there were collections of histiocytic cells in which large empty spherical globules were observed. Nests of lymphocytes were not uncommon; otherwise, there was little or no leukocytic reaction.

Diagnosis.—The diagnosis was chronic fibrosing aspiration pneumonia (lipid, possibly mineral oil).

Discussion (Dr. E. L. Jungherr, Conn.).—I found numerous double refractile bodies in the areas of fibrosis. These suggested silicon and thus the possibility of silicotic fibrosis also occurring in this case.

Dr. J. R. M. Innes (Md.).—I have seen lipid pneumonia in a cow and in a cat caused by transport of fat from elsewhere in the body.

Dr. L. Z. Saunders (N.Y.).—It is common procedure for cat owners to give mineral oil to eliminate hair balls.

Dr. C. L. Davis (Colo.).—The same thing may be seen in mink, and vascular transportation of fat from body deposits should be considered when lesions of this type are seen.

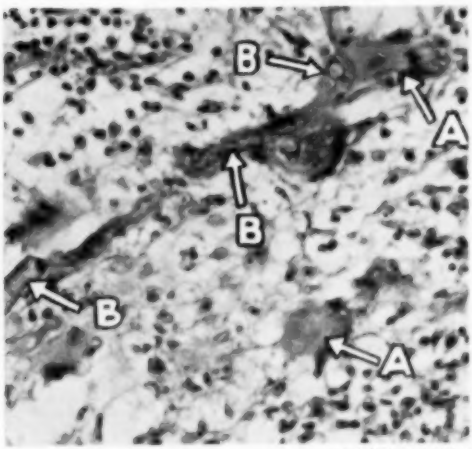
CASE 7—MUCORMYCOSIS IN A STEER—*C. L. Davis, D.V.M.*

History.—This lesion was found on routine postmortem inspection at slaughter in a mesenteric lymph node of an 18-month-old steer in good physical condition. The lesion was 15 cm. in diameter and was irregularly oval with surface lobulations. The cut surface showed a greenish yellow caseocalcareous structure resembling tuberculosis.

Comments (Moderator Jones).—Large areas of a section of the lymph node were refractory to stain. Some areas stained deeply with eosin; others contained necrotic debris in which the outline of macrophages, giant cells, and spicules of calcareous material could be discerned. Toward the periphery, the tissue was better preserved (fig. 8) and there was an

Case 6 was presented by Dr. W. A. Anderson of the Branch Laboratory, Agricultural Research Service, U. S. Department of Agriculture, Denver Federal Center, Denver, Colo.

Case 7 was presented by Dr. C. L. Davis of the Branch Pathological Laboratory, Agricultural Research Service, U. S. Department of Agriculture, Denver Federal Center, Denver, Colo.



—A.F.I.P.—54-22025

Fig. 8—Section of lymph node (case 7) showing hyphae of Mucor organisms (B); also two giant cells (A). x 220.

intense granulomatous reaction which displaced much of the lymphoid structures. The organism, an irregular spherical body which frequently showed budding, could be seen not only in the giant cells but in the granulomatous tissue and in the areas of necrosis (fig. 8). The spherical portions of the organisms varied from 10 to 20 μ in diameter and often sent out irregular branching hyphae. The hyphae were usually less than 10 μ wide but might be as much as 80 μ in length.

Diagnosis.—The diagnosis was mucormycosis of a lymph node.

Congenital Atresia of the Colon in a Calf

CARL F. SCHLOTTHAUER, D.V.M.

Rochester, Minnesota

Congenital atresia of the intestinal tract has been observed in various species of animals, but there is no statistical data available to indicate its incidence. Imperforate anus is the most frequently observed of these anomalies. This, however, probably is because an imperforate anus can be diagnosed by a simple physical examination. When atresia occurs in other parts of the intestinal tract, its site commonly is not determined, because conditions resembling constipation in newborn animals are usually treated by the owner. If the animal dies, necropsy is omitted in most instances and the true nature of the condition present is not learned.

The following case is reported because it is unusual and it illustrates the importance of necropsy when the animal has not responded to treatment. Without a necropsy in this instance, the diagnosis would have been incorrectly recorded as obstinate constipation.

Report of Case

A male Holstein-Friesian calf was born spontaneously at term and was thought to be normal. It was permitted to nurse naturally for two days and then was fed milk from a pail. The owner noticed that the calf did not eat well and that the only semblance of fecal matter passed was some bloody, mucus-like material. Treatment for constipation was ineffective. After this treatment, the calf had slightly labored breathing, refused to eat, and died on the eighth day.

Necropsy disclosed that both lungs were congested, which accounted for the respiratory difficulty. The heart appeared normal on gross examination. The stomach and small intestines appeared normal but the colon was distended and filled with ingesta; however, the colon was not discolored. Further examination disclosed that there was complete atresia of the colon in its terminal portion. The portion of colon distal to the site of atresia, along with the rectum, was 44 inches long. This portion of the intestinal tract was small in caliber and contained only thick, tenacious mucus.

A correct diagnosis of the condition while the animal was alive could have been made only by surgical exploration of the abdomen. The only successful treatment, too, would have been surgical anastomosis of the colon proximal and distal to the site of the atresia.

From the Section of Veterinary Medicine, Mayo Clinic and Mayo Foundation (a part of the Graduate School, University of Minnesota), Rochester, Minn.

Nicarbazin for Coccidiosis.—As a prophylactic against coccidiosis in chickens, nicarbazin fed continuously at a level of 0.02 per cent of the ration was economical and efficient. However, there was evidence of toxicity at a level of 0.04 per cent.—*Cornell Vet., July, 1955.*

Studies on Infectious Atrophic Rhinitis of Swine

III. Review of Literature

WILLIAM P. SWITZER, D.V.M., Ph.D.

Ames, Iowa

INFECTIOUS atrophic rhinitis has been recognized in Germany for a century and a quarter. Franque (1830)[23] published the first report of this disease, referring to it as *Schnüffelkrankheit* (sniffling disease). He recorded the observations of two veterinarians in the mountainous region of Nassau who noticed that affected swine did not fatten, developed an atrophy of the nasal and ethmoid turbinates, a malformation of the nose and, in severe cases, nasal hemorrhage. It was speculated that short-nosed swine were more susceptible and that rachitic pigs rooting in stony ground might develop this condition. Franque thought it more likely that the condition was hereditary although it spread gradually through the entire herd.

Several articles on this disease were published during the next 75 years, most of them presenting ideas similar to those of Franque, with emphasis on heredity, nutrition, or infection as the cause.

Hering (1842)[37] felt that the facial deformity resulted from rickets and that it could be cured in its early stage by feeding bone salts and cod-liver oil. He cautioned that tuberculosis could cause such lesions.

Schneider (1878)[76] had information that *Schnuffelkrankheit* had been recognized at least 70 to 80 years prior to the time of his writing. He concluded that it was inherited.

Schell (1890)[77] described as *Schnuffelkrankheit* a case of osteosarcoma of the facial bones of a pig. Imminger (1890)[42] likewise described an acute febrile rhinitis of swine. Besnoit (1903)[4] felt that *Schnuffelkrankheit* was due to a lime deficiency and could be cured by feeding lime phosphate.

Koske (1906)[52] isolated *Bacillus pyocyaneus* from the nasal cavities and viscera of young swine dying of acute rhinitis and septicemia. He reproduced acute rhinitis by the intranasal instillation of a culture of this organism. He felt that the term *Schnuffelkrankheit* should be used to designate acute rhinitis due to *B. pyocyaneus*.

According to Hintze (1909)[38] *Schnuffelkrankheit* was related to osteodystrophia fibrosa of other domestic animals. Wirth (1910)[102] likewise concluded that this was osteodystrophia fibrosa or deformans. Ingier (1913)[41] reported active osteogenic tumor tissue in the facial lesions and in the skeleton of the body and limbs. Busolt (1912)[9] expressed the opinion that the condition resembled rickets in man.

Jensen (1916),[46] after reviewing the literature on *snovlesyge* (infectious atrophic rhinitis, sneezing sickness, sniffling disease, *Schnuffelkrankheit*), pointed out that three distinct disease syndromes were included: acute infectious nasal catarrh; malformations such as osteopetrosis, osteomalacia, rickets, or ostitis fibrosa deformans; and the original disease described by Franque. In Denmark, where it was referred to as *snovlesyge*, *nysesyge*, or *snoftesyge*, Bang had collected swine specimens showing this disease syndrome as early as 1880. In 1916, of 4,022 swine heads from a packinghouse, 23 had definite turbinate atrophy. In an experiment using 2 normal 6- to 8-week-old pigs in contact with 2 older, infected animals, no transmission was demonstrated. Jensen reported that no similar disease had been observed among other domestic animals.

Petersen (1925)[66] reported the successful treatment of infectious atrophic rhinitis with potassium iodide. Petersen (1926)[67] mentioned that this disease was seen in young pigs, and occasionally as a central nervous system involvement associated with nasal catarrh in older pigs. He concluded that it was a transmissible, enzootic infection, but that there was a variation in the extent of spread and virulence of the disease from year to year. He considered Lugol's solution to be a satisfactory treatment.

Manninger (1930)[55] suggested that the term *ansteckender Nasenkatarrh* (infectious nasal catarrh) was preferable to *Schnuffelkrankheit*. He described it as an acute hemorrhagic rhinitis from which Poels had recovered Pasteurella sp.

Jensen (1933)[47] by now believed that infectious atrophic rhinitis was an infectious disease, since 5 to 10 per cent of the swine in abattoirs in Denmark showed some turbinate damage. This article was abstracted by Biester (1935) and was the first information on infectious atrophic rhinitis to be published in the English language.

Eber and Meyn (1934)[27] recovered *B. pyocyaneus* from an animal with acute rhinitis and reproduced the condition when toxin from this culture was placed in the ethmoid turbinates through a trephined opening. They reported favorable results from immunizing this herd with *B. pyocyaneus* bacterin.

Zarickij (1934)[107] believed that rhinitis of pigs

From the Veterinary Medical Research Institute, Iowa State College, Ames.

This material is based on a dissertation submitted to the Graduate College, Iowa State College, in partial fulfillment of the requirements of the Doctor of Philosophy degree.

in France and rhinoscleroma of man were both due to *Bacillus friedlanderi*.

Hoflund (1937)[60] stated that chronic atrophic rhinitis was fairly common in southern and central Sweden. Unable to isolate any bacteria or to transmit the condition, he concluded that it was an inherited defect. He demonstrated that turbinate atrophy could be detected in the living animal by radiographs.

When Thunberg (1937)[80] cited the introduction of the disease by means of an infected boar, Hoflund (1937)[60] cited support for his contention that the condition was inherited.

Krage (1937)[62] felt that the disease was due to a latent infection that manifested itself in animals with an inherited predisposing factor.

Radtke (1938)[72] reported that *Schnuffelkrankheit* occurred only in association with *Ferkelgrippe* (piglet influenza). He found that the Riemser single-house method of raising swine, used to control *Ferkelgrippe*, also controlled *Schnuffelkrankheit*. He also found that the ventral and dorsal turbinates, the ethmoid labyrinth, and the maxillary sinuses are fully developed at birth. The turbinates contain much cartilage at birth, the dorsal and ventral turbinates becoming completely ossified by the fourth week, and the ethmoid turbinate by the eighth week of life.

Radtke observed that the bacterial flora of the nasal cavity of healthy pigs was reasonably constant, with *Bacillus coli*, diplococci, streptococci, and micrococci occurring most regularly; that the numbers of bacteria decreased in the posterior portion of the nasal cavity; that the ethmoid turbinates and the sinus cavities were usually sterile; and that between 60 and 70 per cent of the nasal cavities from healthy swine harbored *Hemophilus suis*. He examined 52 *Ferkelgrippe*-free pigs 4 to 8 weeks of age and found no rhinitis. He then examined 104 *Ferkelgrippe*-positive pigs from 50 premises and found 39 with rhinitis, the more advanced cases of which had turbinate atrophy. He observed that short-nosed breeds of swine tended to have more severe lesions of *Schnuffelkrankheit* than long-nosed breeds.

Radtke also found that intranasal inoculation of nasal exudate from pigs with *Schnuffelkrankheit* into susceptible pigs would produce bronchial pneumonia typical of *Ferkelgrippe*, and that filtrates (cotton filter) of typical *Ferkelgrippe* bronchial pneumonia would produce nasal lesions similar to the early lesions of *Schnuffelkrankheit*. He concluded that *Schnuffelkrankheit* was a localization of *Ferkelgrippe* in the upper respiratory tract. This is the first report of the successful experimental transmission of infectious atrophic rhinitis.

Thunberg and Carlström (1940)[100] found infectious atrophic rhinitis associated with certain premises. When litters from healthy sows were divided, the pigs placed in a healthy herd remained healthy while those placed in a diseased herd developed the disease. They felt that mature sows exposed to the disease developed a transient infection and would not infect subsequent litters.

Böttcher (1941)[6] suggested that swine from infected herds should not be used as breeding stock. Reinboth (1940)[74] concurred with Radtke's opinion that *Schnuffelkrankheit* was a localization of *Ferkelgrippe*.

A condition that may have been infectious atrophic rhinitis was reported in the United States by York (1941)[102] in an Indiana herd.

Doyle, Donham, and Hutchings (1944)[15] observed the occurrence of a dystrophic or atrophic rhinitis in five herds over a three-year period, it having existed in one small area of Indiana for 20 to 25 years. They felt that the condition was similar to the European chronic atrophic rhinitis, but that it was different from bullnose.

Isa (1944)[54] reported the occurrence of infectious atrophic rhinitis in pigs in Manitoba, Saskatchewan, and Alberta, Canada. Since he felt that it was not practical to attempt to distinguish between this disease and bullnose, he discussed them as one.

Connell (1945)[10] stated that a peculiar type of bullnose had been present in the prairie provinces of Canada for a number of years but that it was distinctly different from the condition produced by *Actinomyces necrophorus*.

Phillips (1946)[69] stated that Kernkamp had observed infectious atrophic rhinitis in Minnesota; also that it had been observed in Ontario, Canada, for about three years. Most of the infected animals had a black area below the eyes which was attributed to occlusion of the tear duct with resultant spilling of tears, creating a moist area that trapped dirt and dust. He found that the microscopic change consisted of a progressive dissolution of the softer bony structures of the nose, an early inflammation of the nasal mucosa being followed by decalcification of the turbinate bones. The occurrence of an encephalitis in a small fraction of the infected pigs, due to entrance of organisms through the damaged cribiform plate, was observed.

Phillips isolated *Corynebacterium pyogenes* from the nasal cavities in over 90 per cent of the cases and concluded that it was the most important secondary invader. Since filtrate from nasal material of infected swine produced typical lesions when instilled intranasally into 5-day-old pigs, he felt that the primary etiological agent was a virus.

Slagsvold (1946)[87] observed an influenza-like disease of young pigs associated with infectious atrophic rhinitis with evidence indicating a common etiology for the two conditions. Infectious atrophic rhinitis was made a quarantinable disease in Norway in September, 1946.

Duthie (1947)[16] failed to transmit the disease to 6- to 12-week-old pigs by contact or to 4-day-old to 3-month-old pigs by intranasal inoculations.

Moynihan (1947)[60] likewise failed to produce the disease in pigs 10 to 12 weeks of age.

Jones (1947)[40] observed that cats kept on infected premises often developed a purulent rhinitis and occasionally a conjunctivitis. However, 6-week-old kittens, guinea pigs, and rabbits were found to be refractive to infection with this disease. When placed in contact with infected swine, a litter of week-old pigs developed the disease while 9-week-old pigs did not.

Jones found that bacteria-free filtrates from nasal exudate of infected pigs failed to reproduce the disease; also that convalescent serum, Corynebacterium sp. hyperimmune serum, or Corynebacterium sp. toxoid failed to alter the course of natural outbreaks.

Jones (1947)[40] attributed some of the loss of condition in pigs affected with this disease to toxemia. He recognized that affected litters were potential carriers but observed that, if sows from an infected herd with no clinical evidence of the disease were moved to noninfected premises, and were farrowed in isolated lots, they did not transmit the disease to their pigs.

Sippel *et al.* (1947)[56] reported the occurrence of infectious atrophic rhinitis in Georgia. Sandstedt (1948)[53] noted its infectious nature but decided that it did not cause the unthriftiness in the infected pigs that swine pneumonia did. He always found it associated with swine pneumonia and felt that this suggested a common etiology for the two conditions.

Schofield (1948)[55] found that the initial reaction was an infiltration of large lymphocytes into the stroma of the turbinate epithelium. The cells of the nasal epithelium appeared elongated or cuboidal and did not become stratified or squamous as in primary atrophic rhinitis in man. Later, there was an increase in the number of tubuloalveolar glands, accompanied by a mild proliferation of the fibrous tissue elements of the stroma. Proliferation of the osteoblasts occurred even when the bone damage was scarcely perceptible, and in advanced cases the osteoblasts were present in enormous numbers. This was interpreted as indicating these cells were putting forth an unsuccessful effort to rebuild the destroyed bone. He felt that the disappearance of the bony plates of the turbinates was the most outstanding characteristic of this disease.

MacNabb (1948)[42, 43] and his group reported that this was a transmissible disease, that no bacteria had been isolated that could be considered as having etiological significance, that filtrates (type of filter not given) failed to reproduce the disease, and that there was no difference in the susceptibility of different breeds of swine to this disease. They found no significant correlation between the length of the pig's nose and susceptibility to infectious atrophic rhinitis. MacNabb reported the successful transmission of infectious atrophic rhinitis by (1) inoculating crude atrophic turbinate material subcutaneously into a rabbit, then (2) instilling material from the resulting abscess in the rabbit into the nasal cavities of experimental pigs.

Bennett (1948)[3] made the first definite diagnosis of infectious atrophic rhinitis in swine in Iowa.

Phillips *et al.* (1948)[51] produced typical lesions in baby pigs by intranasal inoculation with crude infected turbinate suspensions but not with bacterial cultures recovered from atrophic turbinates

Gendreau (1948)[24] exposed both inbred and noninbred pigs and both groups developed typical lesions. He observed that 7- to 8-week-old pigs acquired the infection when placed in a pen which had previously contained infected pigs. Cohrs (1949)[12] agreed with Radtke that it was merely a localization of *Ferkelgrippe*.

Gwatkin *et al.* (1949)[25] found that intranasal inoculation of day-old pigs with nasal washings from infected pigs produced turbinate atrophy. Filtrates (Seitz filter) failed to produce any lesions in day-old pigs but those prepared with a coarse Mandler filter may have caused some turbinate atrophy in 1 pig. They could freeze the nasal washings at -25 F., thaw them, and produce turbinate atrophy. When they inoculated infectious nasal material into the yolk sac and the chorioallantoic sac of chicken embryos, the fourth and sixth chicken embryo-passage material was not infectious for pigs. No embryo lesions were described. Stains prepared from the nasal turbinate material failed to show any rickettsias. The pH of the nasal washings was found to range from 7.34 to 7.51.

Gilman (1949)[28] cited McKay's unpublished experiments as indicating that *A. necrophorus* and *Pasteurella multocida* acted synergistically to produce turbinate atrophy. He found no indication that long-nosed swine were more resistant to this disease than short-nosed swine.

Gwatkin and Plummer (1949),[27] attempting to find a susceptible laboratory animal, instilled nasal washings and filtrates from infected pigs intranasally into mature mice, baby mice, rats, hamsters, guinea pigs, and rabbits but observed no lesions. They also injected nasal washings close to the metatarsal bones of chicks and observed no bone destruction.

Schofield and Jones (1950)[59] reported that recently farrowed pigs, infected by inoculation, readily transmitted the disease to litter mates. Instillation of feces and urine from infected pigs failed to transmit the infection.

Doyle (1950)[14] mentioned that pigs 10 weeks of age developed clinical evidence of the disease after their introduction into an infected herd. Ellison (1951)[20] credits Twiehaus with the statement that this disease had put 60 per cent of the Ontario pork-producers out of business. He felt that the basic lesion in this disease resulted from a failure of the calcium salts to precipitate in the osteoid tissue. Some of the infected pigs which he observed had an increase in the length of the snout. An editorial appearing in *Jen-Sal Journal* (1951)[48] stated that this is one of the most serious diseases of swine and is present throughout most sections of the United States.

Gwatkin *et al.* (1951)[26] observed that anemia

had no effect on the development of experimental cases of the disease, and that the abscess material from rabbits (MacNabb[62, 63]) did not produce turbinate atrophy in young pigs. Filtrates prepared from infective material (Seitz and Mandler filters) proved to be noninfective for young pigs. Crude atrophic turbinate suspensions heated at 45 C. for one hour produced the disease, although heating at 65 C. for one hour destroyed the infectivity of the material. Approximately 2,400 μg. of streptomycin and 3,300 I.U. of penicillin per milliliter of material added to the inoculum 30 minutes prior to inoculation prevented development of lesions in inoculated pigs. A combination of supernatant and washed sediment obtained by centrifugation at 12,000 to 20,000 r.p.m. was more infective than either material alone.

Switzer (1951)[64] was the first to report the occurrence of Trichomonas sp. in the nasal cavities of swine with infectious atrophic rhinitis. He found 70 of 87 affected swine in Iowa (80%) harboring this protozoon in their nasal cavities, while only 2 of 72 swine (2.8%) with grossly normal nasal cavities had this protozoon present. This protozoon did not become established when instilled into the nasal cavities of normal baby pigs, but it did become established in the bovine vagina.

Bennett (1951)[3] reported that of 142 pigs over 3 weeks of age submitted to a diagnostic laboratory during a six-week period, 59 (41.5%) had gross lesions of infectious atrophic rhinitis.

Hutchings (1951)[31] postulated that a virus might be the cause of infectious atrophic rhinitis. It was his impression that it usually required three years for the disease to build up in a herd to the point where it could be recognized. To control this disease he suggested a complete depopulation of swine, disinfection of the premises, and restocking a few months later with pigs free of the disease.

Montgomery (1952)[41] stated that some herds had sent 800 pigs to the rendering plant due to this disease. He stressed the fact that infectious atrophic rhinitis could put swine producers out of business.

Spear (1952)[60] wrote of the danger to the swine industry occasioned by this disease. He noted that the condition was rapidly spreading in Iowa.

Kernkamp (1952)[33] observed that affected pigs sometimes took twice as much feed per pound of gain as normal pigs. He cautioned that pulmonary and gastrointestinal disturbances were not especially characteristic of this disease.

Jones (1952)[30] found that anti-*C. pyogenes* serum and bacto-toxoid failed to control a natural outbreak of infectious atrophic rhinitis and that anti-*P. multocida* serum and bacterin were of no value in controlling this infection. However, an intramuscular injection of 10,000 μg. of streptomycin given three times during the first month after birth appeared to reduce the incidence of the disease.

Spear (1952)[61] observed that the majority of infected pigs became rough in appearance and unthrifty, and that in some herds this infection was eliminated by early recognition and destruction of infected animals.

Simms (1952)[54] reported that workers in the Bureau of Animal Industry had observed that nasal trichomonads were often associated with infectious atrophic rhinitis. He suggested that these organisms were probably involved in one way or another in the production of this disease. Gwatkin and Dzenis (1952)[26] found that 100,000 μg. of streptomycin administered intranasally seven days after the first inoculation appeared to protect 10 of 12 pigs inoculated intranasally with infectious atrophic rhinitis material.

Gray (1952)[23] found that a drove of swine could be infected with this disease, yet show no symptoms. He stressed the fact that this disease built up in herds of swine, reaching its peak about the third year.

Earl and Shuman (1953)[15] found that an otoscope could be used to determine the degree of turbinate destruction present in many infected pigs.

Shuman *et al.* (1953)[52] found in one herd that 60 per cent of the pigs with turbinate atrophy had pneumonia, while 50 per cent of those with grossly normal turbinates also had pneumonia. They obtained no evidence that swine influenza virus was concerned in the production of either this disease or the swine pneumonia they observed.

Shuman and Earl (1953)[52] attempted to evaluate the accuracy of rhinoscopic detection of turbinate damage. They could detect by rhinoscopic examination about 75 per cent of the cases that exhibited turbinate atrophy at necropsy.

Aitken (1953)[1] suggested that a course of treatment with sulfapyridine would differentiate infectious atrophic rhinitis from bullnose. If the pigs recovered, it was assumed they had bullnose.

Dykstra (1953)[17] believed that this disease always produced complete destruction of the turbinate bones if allowed to progress and that an immunity was not developed. Smiley (1953)[50] presented a detailed account of how this disease had built up in a purebred herd and was disseminated to other herds through the sale of breeding stock.

Borgmann (1953)[5] demonstrated that *Ery. rhusiopathiae* was not the cause of infectious atrophic rhinitis as had been suggested by Messmore (1952)[40, 41] and did not appear to have a synergistic action in the production of this disease.

Shuman (1953)[51] observed a lower rate of infection in pigs kept on concrete than in pigs put out on pasture. Spindler *et al.* (1953)[55] considered that their results established an etiological relationship between trichomonads and infectious atrophic rhinitis. They felt that trichomonads might be of either nasal or intestinal origin and still produce turbinate atrophy.

An editorial in the *Fort Dodge Bio-Chemic Review* (1953)[22] stated that clinical evidence of this disease in a group of swine offered for sale at a public sales barn should warrant condemnation of the entire group.

Simms (1953)[30] reported that workers in the Bureau of Animal Industry examined several hundred stillborn pigs or pigs dying at a few days of age and found no evidence of turbinate damage whether or not the dam was infected. He also reported no significant difference in weight gain between pigs with normal turbinates and those with atrophic turbinates in two herds studied. Of pigs on pasture, 67.3 per cent were affected while only 27.4 per cent of a similar group of pigs on concrete showed turbinate lesions.

In 1953,[101] the conclusion was made that trichomonad infection was involved in causing infectious atrophic rhinitis. Ray (1953)[78] believed that trichomonads were common secondary invaders in cases of infectious atrophic rhinitis simply because the accumulated pus served as a good culture medium for them. Goldstein (1953)[20] concluded there was insufficient evidence to establish trichomonads as the cause of infectious atrophic rhinitis. He also believed that in some affected animals pneumonia entirely accounted for the unthriftiness.

Gwatkin *et al.* (1953)[39] found *P. multocida* present in the nasal cavities of 38 per cent of the swine with atrophic turbinates and in 16 per cent of normal swine. They inoculated a culture of *P. multocida*, recovered from a diseased nasal cavity, into the nasal cavities of 2 susceptible pigs. One of these pigs had turbinate atrophy when necropsied two months later. When the culture was combined with filtrate (Seitz filter) of atrophic turbinate before inoculation, 2 of 3 inoculated pigs developed turbinate atrophy. When *P. multocida* culture was inoculated intranasally into rabbits, most of them died but 1 lived for two weeks and had turbinate atrophy when necropsied.

Switzer (1953)[94] isolated a filterable agent from the nasal cavity of affected swine. When propagated in embryonating chicken eggs, it appeared to be a pleuropneumonia-like organism (PPLO). The outstanding lesion observed in the inoculated chicken embryos was severe pericarditis. A filtrate of crude atrophic turbinate suspension (Selas No. 02 filter) produced mild turbinate changes in 5 young pigs.

McKay and Carter (1953)[55] observed "L" forms of *Spherophorus necrophorus* in 71 affected pigs, but without the tissue necrosis usually associated with this organism. The reversion of the L forms to bacillary forms, on prolonged incubation and repeated subculturing, was considered as evidence that they originated from *S. necrophorus*.

McKay and Carter (1953)[56] reported that abscess material, produced in rabbits with crude atrophic swine turbinate material, consistently yielded *P. multocida* and L-type colonies of *S. necrophorus* after one or two rabbit passages. However, pure cultures of *P. multocida* failed to produce turbinate atrophy in pigs.

When Switzer (1953)[95] injected a suspected PPLO, isolated from the nasal mucosa of affected swine, intraperitoneally into pigs 6 weeks or less of age, severe fibrinous pericarditis, pleuritis, and peritonitis resembling field cases resulted. He examined 9 such field cases and found 8 of them positive for this PPLO. However, when he instilled this agent into the nasal cavities of 14 baby pigs, no gross atrophy of the nasal turbinate resulted even though the agent became established in the nasal cavity.

Myers (1953)[58, 59] believed that infectious atrophic rhinitis was caused by a nutritional deficiency with secondary bacterial invaders producing the turbinate atrophy.

Carter and McKay (1953)[13] reported that many of the organisms referred to in their previous reports as L-type colonies of *S. necrophorus* were in reality PPLO.

Phillips (1953)[70] expressed the opinion that a synergistic relationship between two agents might still prove to be the cause of infectious atrophic rhinitis.

Smith (1953)[89] found that pigs 4 to 8 weeks of age placed in contact with field cases of infectious atrophic rhinitis did not develop lesions of the disease.

Gwatkin *et al.* (1953)[39] found that a pure culture of *P. multocida* instilled intranasally into young pigs produced nasal changes typical of infectious atrophic rhinitis. Gwatkin and Dzenis (1953)[40] isolated cultures of *P. multocida* from 6 pigs with field cases of infectious atrophic rhinitis and from a pneumonic lung and produced typical lesions in experimental pigs by the intranasal inoculation of these isolates.

Schofield and Robertson (1953)[80] observed turbinate atrophy in baby pigs inoculated with atrophic turbinate material containing no demonstrable trichomonads. No transmission occurred when pens that had contained pigs with infectious atrophic rhinitis were left idle for three weeks before young susceptible pigs were placed in them. They believed that turbinate damage was most likely due to injury to the osteoblasts.

Flatla and Braend (1953)[21] and Braend and Flatla (1954)[7] found that hereditary factors were not concerned in this disease. They initially believed Hemophilus sp. might be an etiological factor but later found it would not produce turbinate atrophy. Cultures of *P. multocida* instilled intranasally produced typical turbinate atrophy. Radiographs assisted them in sorting out infected animals.

Editorials in the *Veterinary Record* (1954)[100, 101] quoted notices issued by the Ministry of Agriculture and Fisheries, of May 20, 1954, that for the first time on record atrophic rhinitis had been confirmed among pigs in Great Britain, doubtless introduced by an apparently healthy pig imported from Sweden. Quarantine and slaughter regulations were established. The *British Veterinary Journal* (1954)[9] expressed gratitude that the officials responsible for animal protection were alert to recognize this new disease.

Switzer (1954)[97] reported recovery of his pre-

viously described PPLO from 20 of 28 pneumonic swine lungs and from the nasal cavities of 14 of these 20 animals. This suggested a correlation between the occurrence of this agent in the nasal cavity and in the pneumonic lung.

Levine *et al.* (1954)[36] observed nasal trichomonads in 10 of 11 pigs with turbinate atrophy and in 9 of 23 pigs with no nasal lesions. They were unable to produce turbinate lesions in 11- to 39-day-old pigs by intranasal inoculation of cultures of nasal trichomonads recovered from field cases.

Carter (1954)[10] found that baby pigs developed no lesions when inoculated intranasally with cultures of the swine PPLO. He isolated this organism from three field outbreaks of serofibrinous pericarditis, pleuritis, and peritonitis in swine. Young pigs inoculated intraperitoneally with these cultures developed lesions similar to those observed in the field cases and the organism was recovered from these lesions.

Heddleston *et al.* (1954)[20] reported isolation of *P. multocida* from the nasal cavities of 6 of 76 swine (8%) with infectious atrophic rhinitis and from the nasal cavities of 4 of 96 rhinitis-negative swine (4.3%).

Switzer (1954)[40] presented evidence indicating that *S. necrophorus, P. multocida,* and nasal trichomonads were not concerned in the production of turbinate atrophy in an isolated herd of infected swine. Selas No. 02 filters did not allow the atrophy-producing agent to pass. However, one pneumonia specimen, from a pig with turbinate atrophy, yielded no bacterial growth yet produced a definite turbinate atrophy in baby pigs. He reported the cultivation in artificial medium of the filterable agent he had previously isolated. It was a typical PPLO. He outlined three general plans for the control of this disease, the most effective of which was to take the pigs from the dam when they were 24 hours or less of age, and to raise them in isolation.

Braend and Flatla (1954)[7] suggested that the most effective method of controlling this disease is complete depopulation of the swine on an infected farm followed by restocking with normal swine. Another method is to isolate the bred sows, to keep the litters in isolation for five months, and to select breeding stock from the litters that then appear normal. These workers also reported that local and systemic treatment of the sows with streptomycin was an aid in eliminating the infection, but they cautioned that the use of antibiotics might result in resistant strains of organisms.

Gwatkin *et al.* (1954)[21] reported that Selas No. 02 filter did not allow the atrophy-producing factor(s) to pass. However, they again demonstrated that cultures of *P. multocida* isolated from field cases would produce turbinate atrophy when inoculated into suitable baby pigs and that these pigs would transmit the condition to uninoculated pigs. They found that broth cultures of the swine PPLO produced a rhinitis in some of the inoculated baby pigs but only slight evidence of turbinate atrophy. It was shown that the nasal material from the eighth rabbit passage of crude swine turbinate material still produced moderate atrophy when inoculated intranasally into baby pigs. This rabbit material contained *P. multocida.* No immunity to turbinate atrophy was evident in pigs vaccinated ten to 14 days previously with *P. multocida* bacterin.

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Chicks React to Foot-and-Mouth Virus

It had been believed that birds were resistant to the virus of foot-and-mouth disease. However, in April, 1953, when the eighty-fifth mouse passage of an O type strain was injected intramuscularly in chicks a few hours old it was recovered from the muscle and the heart four days later. During a series of 12 ultimate passages in chicks and mice, this virus could be readily recovered from the blood of the chicks three to five days after inoculation. When newly hatched chicks were given the original strain of virus intramuscularly, tongue lesions, often involving the epithelium of the entire dorsum as in other susceptible species, developed in one to two days. Other strains of virus would cause a separation of the deeper epithelial layers on the ventral surface of the feet of chicks, with severe secondary lesions on the tongue developing later. No severe systemic disturbances were observed.

Virus from a ninth alternate mouse-chick passage, when inoculated intravenously into 14-day-old chicken embryos, multiplied chiefly in the myocardium with the highest infectivity developing in five to six days. When incubated at temperatures above 35 C., the chicken embryos develop macroscopic lesions in the cardiac muscle and usually die in three to six days.

When the virus of vesicular stomatitis is inoculated intradermally into the tongue, both young and adult birds are highly susceptible, developing severe lesions within 24 hours. Two ducks similarly inoculated also developed lesions.—*Nature, Dec. 4, 1954.*

The screwworm infestation of livestock in Texas in the summer of 1955 has been much greater than usual.—*U.S.D.A., Aug. 5, 1955.*

Only 2 egg-laying mammals are known, the echidna, a porcupine-like animal and the duck-billed platypus, both of which come from Australia.—*Sci. News Letter, Feb. 12, 1955.*

Oxytetracycline in Bovine Mastitis

A study was made to evaluate the use of oxytetracycline (terramycin®) in infectious bovine mastitis. Treatment of 115 animals, clinical and subclinical, with repeated 400-mg. doses of commercial infusion ointment showed the following rates of recovery: streptococci, 82.7 per cent; *Staphylococcus aureus*, 25.0 per cent; coliform organisms, 69.3 per cent; and *Pseudomonas aeruginosa* 0.0 per cent. Results with subclinical mastitis were better than with clinical cases. Results were also better in animals with recently acquired rather than those with long-standing infections, especially those caused by *Staph. aureus*. Use of oxytetracycline, intramuscularly, routinely aided in the relief of general symptoms accompanying mastitis, irrespective of the etiological agent.

Oxytetracycline was found to be irritating in certain instances. Doses of 2 Gm. of infusion ointment intramammarily and 5 Gm. of the hydrochloride salt intramuscularly produced marked inflammatory reactions. Treatment of both normal and infected quarters with either one or three 400-mg. doses of infusion ointment resulted in variable inflammatory reactions as measured by laboratory tests. The *in vitro* sensitivity to the lethal and inhibitory effects of oxytetracycline was determined for strains of *Streptococcus uberis* and *Staph. aureus*. Both were inhibited by approximately 1 μg. per milliter but nine of 12 strains of *Staph. aureus* were killed only by concentrations greater than 200 μg. per milliter, while seven of nine strains

of *Str. uberis* were killed by less than 10 μg. per milliter. If *Staph. aureus* must be killed to eliminate the infection, this refractiveness could partially explain the low recovery rate in this work.—*Am. J. Vet. Res., July, 1955.*

The Bardex Tube as an Aid to Radiography of the Large Bowel in the Dog

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College Station, Texas

The Bardex tube is a rubber catheter (fig. 1) with an air-tight circular cuff that will expand when inflated. The tube is especially useful in radiography because the catheter can be secured in the posterior bowel by placing the end and cuff inside the anal sphincter. About 100 cc. of air seals the anal sphincter and keeps the tube in place and at the same time allows for gas or solutions to be introduced and maintained in the large bowel while radiographs are taken.

Fig. 1—Bardex tube showing inflated cuff and inflation bulb.

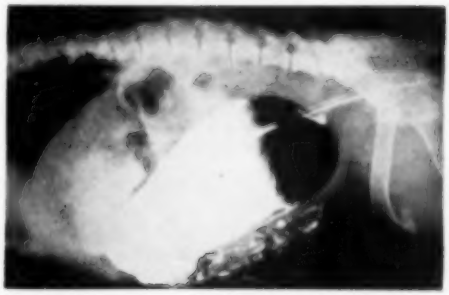

Fig. 2—Lateral view of Bulldog, showing colon being filled with barium solution.

Figures 2 and 3 are radiographs of a 6-year-old female English Bulldog, showing the colon being filled with barium solution. This dog had suffered from chronic constipation since birth and for the past year the condition had worsened to the point that it was necessary to assist the evacuation of the bowel by the means of enemas at monthly intervals.

After the bowel was evacuated, the introduction of a barium solution by the aid of the Bardex tube showed the condition to be a congenital megacolon.

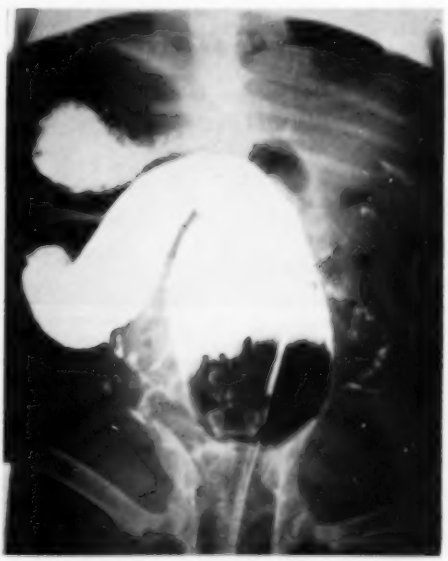

Fig. 3—Dorsal view of the barium-filled colon of the Bulldog.

Chronic Erysipelas-Arthritis in Swine

(An Abstract)

DENNIS SIKES, D.V.M., Ph.D.; GEORGE M. NEHER, Ph.D.; L. P. DOYLE, D.V.M., Ph.D.

Lafayette, Indiana

A total of 248 swine were used in this study. Eight were naturally infected chronic cases of swine erysipelas and 240 were experimentally inoculated. Many of the latter did not develop infection.

Fig. 1 (Fig. 6)—Kidney from a shoat with chronic erysipelas, showing bluish areas caused by infarcts (the "marbled" effect frequently observed in acute field cases).

None of 15 shoats showed signs of infection when inoculated four times at intervals of one to three weeks, subcutaneously, intramuscularly, or intravenously with a rough culture of *Erysipelothrix rhusiopathiae* isolated from the joints of a naturally infected hog. Two animals did

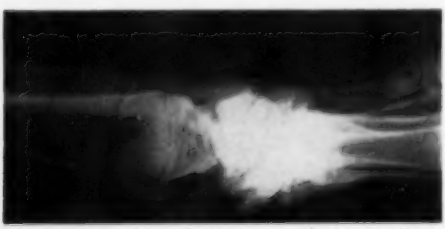

Fig. 2—Radiogram of the carpus (metacarpals on right) of a hog with chronic erysipelas-arthritis showing ankylosis and osteophyte formation.

have a mild fever for two days after inoculation. These apparently healthy shoats may have developed resistance from previous exposure. However, when shoats from erysipelas-free herds were inoculated with smooth colonies of *Ery. rhusiopathiae*, cultured in a 10 per cent horse serum mediums, many became infected.

When 31 were given a single large intravenous inoculation, 18 died within six days with acute erysipelas, 6 developed chronic erysipelas-arthritis, and 7 showed not even a febrile reaction. Two of the arthritic pigs recovered in about two months. Of 16 uninoculated shoats kept in contact with this group, 3 died of acute erysipelas on the seventh or eighth days, 3 developed chronic arthritis, and 10 remained healthy. The erysipelas organism was recovered from all of these acute cases and from the joints of 6 of the 7 shoats with chronic arthritis.

When 13 unexposed shoats were inoculated by the skin-scarification method, 3 died in eight days and 4 developed chronic arthritis. All 13 had marked local reactions but 6 showed no further signs of infection. Of the 6 contact shoats, which were scarified but not inoculated, all had skin reactions — 2 died in eight days, 1 developed chronic arthritis, and 3 remained healthy.

When the erysipelas organism was injected intra-articularly into one carpal and

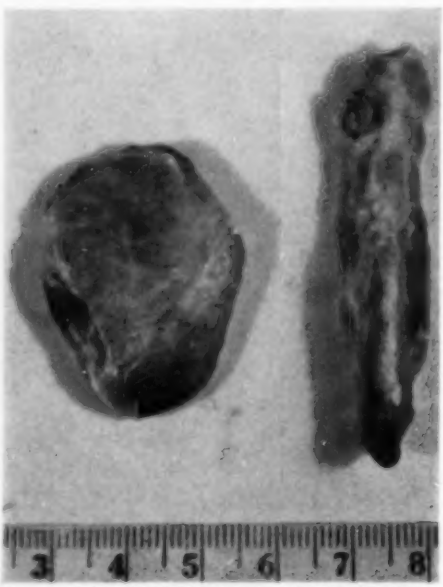

Fig. 3—Adrenal glands from a hog with chronic erysipelas-arthritis; abnormal shape (left) and normal shape (right). The adrenal glands were usually enlarged, often to twice normal size, in chronic cases.

The original article appeared in the American Journal of Veterinary Research, 16, (July, 1955): 349-366.

one tarsal joint in each of 29 shoats, 3 died of acute erysipelas in seven to 14 days while 4 died and 1 was moribund with acute arthritis between 24 and 54 days, and 1 died with a vegetative endocarditis at 112 days postinoculation. On necropsy of the 4 arthritic shoats, no difference could be detected in the inoculated and corresponding uninoculated joints, and the organism was recovered from all of these joints. Of the remaining 20 shoats, 11 developed chronic arthritis (1 recovered in 6 months), 5 showed illness without arthritis but recovered in 60 to 120 days, while 4 remained healthy.

Of the 20 uninoculated shoats in contact with this group, 5 died of generalized erysipelas within 16 days, 4 developed chronic arthritis, and 11 remained healthy.

When 18 shoats on alfalfa pasture were inoculated, 6 intramuscularly, 6 subcutaneously, and 6 intra-articularly, 3 in each group remained well, 5 developed chronic polyarthritis, and 2 in each of the subcutaneously and intra-articularly inoculated groups died of acute erysipelas. Of 12 contact shoats, 3 died of acute erysipelas, and 1 with vegetative endocarditis died on day 124. Eight remained healthy.

The joints of animals with chronic arthritis seldom yielded *Ery. rhusiopathiae* five months after exposure, yet the pathological processes often persisted with periods of exacerbation and remission.

New Milk Production Record for Goats

A 3-year-old Saanen goat recently set an all-breed record with 4901.1 lb. of milk in a 305-day test period. This is an average of about 1 gal. twice a day. She also set a new breed record with 182.79 lb. of fat. This Arkansas goat was fed 4 lb. of grain daily, with all the beet pulp and hay she would eat.—*Dai. Goat J., March, 1955.*

Fog Paralysis in Cattle of Japan.—For several decades a disease known as "fog paralysis," believed to be due to a poisonous plant, has killed many cattle in a limited region of Japan. The disease is now believed to be due to malnutrition and to be precipitated by an abrupt change in management or weather.—*Vet. Bull., April, 1955.*

Toxicity of Chlordan for Turkeys

When an infestation of ants required the use of an insecticide at the Ohio Agricultural Experiment Station, equal parts of 2 per cent chlordan and 0.4 per cent lindane solution was used as a spray. Experimental turkey poults 6 weeks of age, 11 of which were housed in individual cages, remained in the room while the floor and their cages were exposed to a spray mist. All poults died from 55 to 151 hours after being sprayed. One was removed after 24 hours but died 31 hours later. Three poults, placed in these cages a week after the spraying, also died within 132 hours while 6 others in clean cages placed in the same room remained healthy. Since lindane when used in this manner has not shown toxicity, the chlordan was held reponsible.—*Poult. Sci., May, 1954.*

Induced Bloat in Cattle

Bloat was induced 429 times in 5 dry cows, including 2 pairs of identical twins, which were stall-fed on cut red clover in New Zealand. Bloat was produced at all stages of wilting until the dry matter content reached 72 per cent, and it depended little on the rate or total amount consumed. Neither rumen movement nor eructation were inhibited during the onset of bloat. Foaming seemed to be the cause, and antifoaming agents the only reliable preventive. Treatment with adrenaline and with an antihistaminic aggravated the condition. There seemed to be an inherited tendency to bloat.—*Vet. Bull., July, 1955, from New Zealand Sci. Tech., 36: 289.*

Report on New Synthetic Steroids

Prednisone (meticorten), formerly known as metacortandracin, was given orally to 12 patients with active rheumatoid arthritis, one with gouty arthritis, and to two other patients. The dosage was 30 mg. daily for suppression, then 5 to 20 mg. daily. Optimal benefits achieved were similar to those with cortisone or hydrocortisone but with a fraction of the dosage.—*J.Am.M.A., May 21, 1955.*

Actinomycosis in the lungs of 2 cattle had apparently been introduced by pieces of wire penetrating from the reticulum.—*Vet. Bull., April, 1955.*

Coccidioidomycosis in a Kansas Dog

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Stillwater, Oklahoma

Since the identification of *Coccidioides immitis* infection in man by Wernicke in 1892 in Buenos Aires, and in the ox by Giltner in 1894 in California, many cases have been reported in man and cattle. Most of the reports have been from areas of California and Arizona.

Canine coccidioidomycosis has been reported from Arizona,[1] Quebec,[2] Iowa,[3,7] Texas,[4] and California.[5,6] This present report is believed to be the first from the Kansas-Oklahoma area.

CASE HISTORY

An 18-month-old Brittany Spaniel from Kansas was admitted to our clinic (School of Veterinary Medicine, Oklahoma A. & M. College) Nov. 8, 1954. The dog was born in Johnson County, Kansas, and was taken to Wichita when 7 months old. It had never before been out of Kansas except for two days spent in Bates County, Missouri.

Fig. 1—This antemortem, positive radiograph shows many dark specks throughout the lung of the dog. A radiographic shadow is visible dorsal to the heart (1).

The dog was a chicken killer, so the owner thought that the coughing, loss of appetite, wheezing respiratory sounds, and loss of weight which became apparent when the dog was about 15 months old resulted from the inhalation of feathers. The dog, previously vaccinated for both distemper and rabies, had been treated with penicillin and streptomycin.

Fig. 2—Pulmonary granuloma showing caseation necrosis and calcareous deposit (1). x 50.

DIAGNOSTIC PROCEDURES

A radiograph (fig. 1) demonstrated generalized, chronic, fibrotic pneumonia; consolidation of lung apices; and multiple, miliary shadows scattered throughout the lung fields. There was also a radiographic shadow dorsal to the heart.

Sternal bone marrow smears were normal. The dog was negative to an intradermal bovine tuberculin test. An examination of the blood revealed a leukocyte count of 9,050, with a percentage distribution of 43 segmenters, 30 stab cells, 17 lymphocytes, 7 eosinophils, and 3 monocytes. There were 12 Gm./100 ml. of hemoglobin and an erythrocyte count of 4,350,000. The temperature remained normal.

The clinical, radiological, and laboratory findings suggested a diagnosis of mycotic pulmonary infection.

While anesthetized on November 18, to permit a bronchial swab, the dog died. Necropsy showed a severe congestion of the gastric and small intestinal mucosa; an area of slight fibrosis near the interior

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The authors acknowledge the cooperation of the following staff members of the School of Veterinary Medicine, Oklahoma A. & M. College: Drs. N. B. Tennille, radiology; B. L. Glenn, gross pathology; W. E. Brock, microphotography, and E. L. Blevins, clinical observation.

aspect of the cortex of each kidney, with moderate congestion of the right kidney; and numerous small translucent lesions 4 mm. or less in diameter scattered throughout both lungs. The anterior mediastinal nodes measured about 2.5 cm. by 1.5 cm. and were severely congested. The bronchial nodes (the radiographic shadow) were about 5.5 cm. by 3.5 cm. and contained calcareous deposits. There was one small hemorrhagic area in the spleen and another on its free border.

Tissues from various organs were fixed in 10 per cent formalin, and sections were made and stained with hematoxylin and eosin, Schiff, and carbolfuchsin stains. Many tubercle-like granulomas (fig. 2.) containing *C. immitis* spherules (fig. 3 and 4) were found in the lung tissue.

Cultures from fresh lung and liver tissue were made on bovine blood agar, Sabouraud's dextrose agar, and Stanford diagnostic "K" medium, and growth occurred on all mediums between the forty-eighth hour and the sixth day. The mycotic growths were characteristic of *C. immitis*.

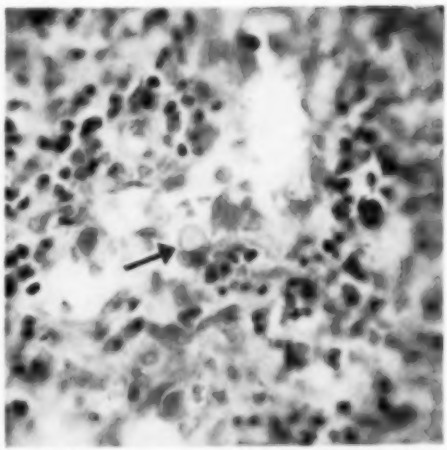

Fig. 3—The arrow points to a phagocyte containing an immature spherule in this bronchial lymph node section. x 490.

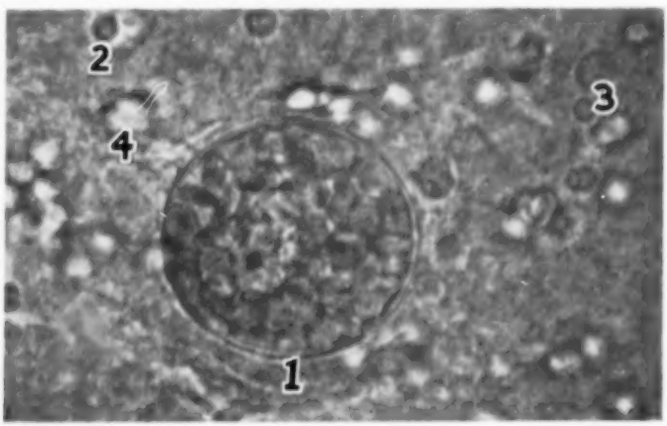

Fig. 4—A giant endospore-filled spherule (1) and immature spherules (2, 3, 4) can be seen throughout the field. x 1,225.

A loopful of arthrospores suspended in 1 ml. of sterile physiological solution was injected into a testicle of a guinea pig which died in 12 days. Growths obtained from cultures of the guinea pig's liver positively identified the organism.

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Oral Metrazol Therapy in Aged Dogs

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THE PRACTICE of a veterinarian who treats only small animals is made up of a much higher percentage of physiologically old patients than is the practice of a physician, unless the physician specializes in geriatrics. This is so because of the short span of years which comprises a canine generation. We do not suggest geriatrics as a veterinary specialty, but we are interested in solutions to the clinical problems presented by aged dogs.

Some time ago, a physician told us of the oral use of metrazol® in human geriatric patients. Since we do not have ready access to the bulk of medical literature, we asked the manufacturers of metrazol (Bilhuber-Knoll Corp.) to supply this information. From it we gained the conviction that this drug should be of as much value to aged dogs as it is to elderly human beings.

Thewlis[1] emphasized that diseases in senility are pathological conditions in normally degenerating bodies and that the object of treatment is to restore the involved organs or tissues to the state of normal in senility but not to the state of normal in presenile maturity. Because of anatomic changes of senility, the functions of organs and tissues are altered, but the functional changes do not always correspond to the anatomic changes. "The capitol physiological changes are: lessened activity, altered activity, and loss of harmonious inter-relation between associated functions. These changes are particularly evident in respect to eyes, ears, teeth, respiratory tract, circulatory system, blood, genitourinary tract, gastrointestinal tract, nervous system, brain, basal metabolism and skin." The respiratory capacity is diminished and the amount of air inhaled tends to be insufficient for adequate oxygenation of the blood. The circulatory system is less able to supply the terminal vessels, so the organs and tissues are often insufficiently nourished. The heart usually is somewhat dilated and hypertrophied and the muscle is weakened, resulting in decreased pulmonary systemic blood flow.

Mason and Schefler,[2] of our own branch of medicine, emphasized some important similarities and differences in senility as exhibited by dogs and by man, as follows: "There is a large literature on the neuropsychiatry and neuropathology of human seniles . . . . Canine and human senility show similar clinical but not neuropathological features . . . . Characteristics are decreased reactivity to stimuli with decreased perception; defects in memory, association, learned behavior, and habit; irritability and anger reactions; snapping at imaginary objects (in the case of dogs); and such neurological signs as posterior weakness (in the dog), tremors and ataxia. . . . These are classic features . . . due to cerebral destruction of cortical neurons. Senile man and dog have these (neuronal) defects in common. . . . It is seen that neuronal loss and gliosis, generally attributed to chronic anoxia, form the common denominator of human and canine senility . . . . the psychiatric changes described will follow exposure to anoxia at any age. . . . In human patients, anoxia-like brain changes have been attributed to decreased vascular supply that follows arterial narrowing due to arteriosclerosis. This explanation will not suffice (in veterinary practice) as arteriosclerosis is not often present in dogs. Possibly reduced cardiac output, anemia, or changes in the nerve cells themselves leading to cellular anoxia are responsible. . . ."

Fazekas and Bessman[3] stated that: "Many disturbances of cerebral function result from metabolic disturbances with the nervous system. . . . At body temperature, enzymes are necessary to catalyze the energy-yielding reactions between substate and oxygen. Any disturbance, therefore, which interferes with either substate supply, enzymatic activity, or oxygen supply of cerebral cells must necessarily result in impaired function. Those cerebral cells with the highest metabolic requirements . . . are first affected by such energy deprivation. . . ."

Houston[4] adds that: "The mental signs of anoxia may be subtle and easily mistaken for primary neurological disease. Mental confusion, emotional lability, errors in judgement, inability to concentrate, headache, and gross tremors are frequently caused by oxygen-lack alone and may be a principal evidence of its presence. Those signs and symptoms are often seen in elderly persons."

During the last few years, metrazol has been widely investigated as a therapeutic agent in the field of human geriatric-psychiatric medicine.[5-10] It is currently being used with success in senile patients who have mental disorders, become unduly fatigued, are anorexic, and who have devel-

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oped bladder and bowel incontinence. Apparently, it facilitates transmission of nervous impulses over the synpatic junctions of cerebral neurons. It also acts to restore normal functional activity to the brain, deepen respirations, improve the circulation, and increase cerebral oxygenation.

Because of its demonstrated benefits to human patients, metrazol would seem to be indicated in many cases in small animal practice. When used on 76 animals, mostly old dogs, the results were encouraging. Table 1 gives the pertinent details of 9 representative cases in which it apparently was beneficial.

Metrazol proved to be more effective than sedative drugs in a dog with idiopathic nocturnal epilepsy and merits further clinical trial in these cases. There seems to be no short explanation of the cause of epileptiform convulsions. However, a neurologist[1] suggests that at least some epileptic episodes, particularly those occurring at night, and those due to hypoxic or physiological (sleep) depression of the cerebral cortex, or to both depressant influences, whose inhibitor effect on the hypothalamus ordinarily prevents this brain structure from becoming overactive and precipitating convulsions. By improving the respiration and circulation this drug helps overcome the cortical depressant effect of cerebral hypoxia. Furthermore, it directly stimulates the cerebral cortex, which is physiologically depressed by sleep, and tends to maintain stronger regulatory nervous impulses from cortex to hypothalamus, thereby acting as an anticonvulsant drug in idiopathic epilepsy.

The drug is also indicated in infectious diseases and toxemias which threaten the lives of animals by causing toxin depression of the cerebral respiratory and vasomotor centers, and where respiratory-circulatory support is needed to sustain vital functions until antibiotics, serums, and other measures can bring the toxemia under control. In infectious diseases, metrazol therapy seems to result in a more rapid return of normal circulation and respiration and a faster return to normal activity and appetite.

In cardiac patients, the simultaneous use of digitalis and metrazol often gives excellent results. The latter indirectly stimulates the medullary respiratory and vasomotor centers, thus complementing the action of digitalis on the heart. According to Wright[2] ". . . a slightly diminished oxygen supply to the heart may impair its efficiency and decrease its output, so that the circulation may become inadequate for the needs of the body. An enfeebled respiratory center, owing to the malnutrition resulting from a failing circulation, is unable to inaugurate (respiratory) movements of adequate depth. The breathing consequently becomes excessively shallow, and dangerous anoxia may develop."

TABLE 1—Results of Metrazol Therapy

Breed	Age	Sex	Tenative diagnosis	Signs and symptoms	Dosage	Remarks
Boston Terrier	10 yr.	F	Senility and generalized lymphosarcoma	Cachexia, drowsiness, dyspnea, cyanosis, enlarged lymph nodes	1.5 gr. t.i.d.	In 15 days, despite the lymphosarcoma, active and alert. Termination apparently delayed.
Cat	17 yr.	F	Senility	Anorexia without evident cause	0.75 gr. t.i.d.	In 3 days, eating, active, euthanasia delayed.
Airedale	15 yr.	M	Senility, partial paraplegia, and chronic nephritis	Could not rise, needed assistance to walk	1.5 gr. t.i.d.	In 5 days, could walk normally; alert, eating well, less polyuria; then died from uremia.
Doberman Pinscher	7 yr.	M	Obesity and partial paraplegia	Overweight, general weakness	1.5 gr. t.i.d.	In 3 days walking almost normally, lost much superfluous weight in 5 months.
Cocker Spaniel	3 yr.	F	Idiopathic epilepsy	Convulsions; recurrent for a month despite dilantin and phenobarbital therapy	1.5 gr. t.i.d. for 102 days	Relieved convulsions for 15 weeks, euthanasia then requested.
Setter	8 yr.	M	Idiopathic syncope	Fainting spells, only	1.5 gr. once daily for 15 mo.	No fainting except when medication stopped for 4 days.
Cocker Spaniel	10 mo.	M	Pneumonia, secondary to distemper	T. 104.6 F., dyspnea, cyanosis, purulent discharge	1.5 gr. t.i.d. & antibiotics for 7 days	Hastened recovery.
Spitz	12 yr.	M	Congestive heart failure	Listless, dyspnea, chronic cough, valvular insufficiency	1.5 gr. t.i.d. & 0.2 mg. digitoxin daily	Little improvement on 0.2 mg. digitoxin for 16 days. General improvement in 2 days when metrazol added.
Toy Collie	9 yr.	M	Delayed recovery from anesthesia	Stuporous, and refused food for 2 days after pentothal	1.5 gr. t.i.d. for 10 days	Markedly hastened recovery from anesthesia.

There are excellent reasons for giving aged, toxic, or debilitated patients a centrally acting respiratory-circulatory-cortical stimulant before operating and during the postoperative recuperative period. Metrazol, given preoperatively, helps protect the medullary centers against undue depression by the anesthetic agent and unavoidable surgical trauma. Stieglitz,[10] emphasizes that the brains of elderly persons anesthetize easily and they tend to remain asleep too long. Postoperative metrazol medication acts to prevent this. It also largely eliminates increased venous stasis which is primarily responsible for postoperative thrombosis and embolism, and it improves pulmonary ventilation and voluntary coughing, which guard against the development of atelectasis. Mobility, plus efficient breathing and vigorous circulation, increases the supply of blood, oxygen, and nutrients to the tissues and increases elimination of metabolites, while respiratory-circulatory inadequacy, anoxia, and hypoproteinemia retard healing and recuperation.

We have had several patients that tended to remain in a half-anesthetized state too long after surgery. They refused food and water, would lie almost motionless, and their breathing was slow and shallow. Metrazol orally was effective in quickly restoring these animals to normal activity. Orally, usually one tablet three times daily, it has also been of considerable benefit to 76 patients, 26 of which presented evidence of senility as reflected by disturbances of the central nervous system, the respiratory system, and the cardiovascular-renal system. Most of the remaining 50 animals were aged, toxic, debilitated, and poor risks as surgical patients.

Usually, the most clearly manifested, clinically recognizable signs of senility in the dog derive from reduced functional capacities of the nervous system, especially the brain, and from the pulmonary and cardiovascular-renal systems. The rapid, shallow, and often difficult breathing and the sluggish and inefficient circulation of senile dogs make for decreased oxygen supply to all body tissues. This hypoxia is particularly disturbing to the brain and heart muscle. The 26 animals which were benefited by metrazol exhibited lassitude, malaise, anorexia, congestive heart failure, cardiac asthma, pneumonia, paraplegia, facial paralysis, idiopathic syncope, and nocturnal epilepsy.

The 50 surgical patients that were given metrazol orally were aged, toxic, weak, or otherwise debilitated. The drug was used only limitedly as a preanesthetic medication to support the physiologically or toxin-depressed respiratory and vasomotor centers of these poor-risk patients. However, we feel it should be used more routinely for this purpose, particularly as it had no appreciable effect on the amount of anesthetic required or on the anesthetic state. As postoperative medication for aged, poor-risk surgical patients, a 1-cc. injection of metrazol at the conclusion of the operation, and one tablet given orally three times daily during periods of 12 to 72 hours following operation, showed excellent results.

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Scours in baby calves, when chiefly due to coliform organisms, responds best to chloromycetin,® 750 mg. orally, followed by 500 mg. in twelve hours if necessary.—*G. R. Moore, D.V.M., Michigan.*

Corneal Inoculation of Rabies.—Encephalitis developed with considerable regularity in the six to eight days following corneal inoculation of rabbits with fixed rabies virus.—*Vet. Bull., July, 1955.*

What Is Your Diagnosis?

Because of the interest in veterinary radiology, the JOURNAL publishes this month, and will continue to do so for the next several issues, a case history and accompanying radiographs depicting a diagnostic problem.

Make your diagnosis from the picture below—then turn the page ▸

History.—A Thoroughbred gelding, 9 years old, had been treated for one year for an enlargement of the left carpus. A radiograph (fig. 1) of the joint was taken for an evaluation of the pathological changes. The owner wanted an opinion on the extent of the continued disability to be expected.

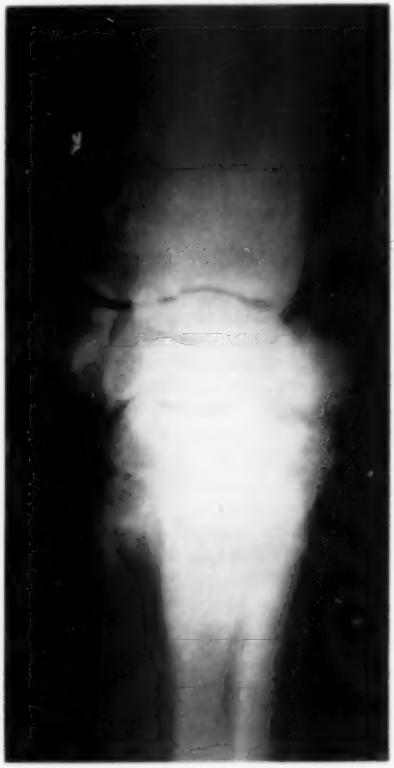

Figure 1

(Diagnosis and findings are reported on next page)

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Hypertrophic arthritis.

Comment.—The prognosis was unfavorable with little hope for successful treatment. The lesion restricted movement to 25 per cent of normal. It is of interest that massive doses of cortisone injected directly into the joint capsule enabled this horse to race in 1954, although his racing record was not good.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

This case was submitted by Drs. John P. Manning, Harry Hardenbrook, and Lloyd E. Boley, Department of Clinics, School of Veterinary Medicine, University of Illinois, Urbana.

Erysipelas in Saskatchewan Turkeys

Erysipelas in turkeys was recognized for the first time in Saskatchewan in January, 1955, in two flocks 100 miles apart. The birds affected were mostly young males. Anti-swine erysipelas serum, 20 to 30 ml. per bird, gave satisfactory results both in prevention and treatment. Aside from depression, the first sign was petechiae on the caruncles and on the skin of the breast. The lesions often coalesced into a hemorrhagic area with the skin becoming necrotic and sloughing. In those necropsied, the only constant lesion was an enlarged spleen which regularly yielded *Erysipelothrix rhusiopathiae.*—*Canad. J. Comp. Med., May, 1955.*

Lungworms in Pigs and Guinea Pigs

Of 1,308 slaughtered pigs in Britain, 18 per cent were affected with Metastrongylus in the lungs. Of 116 guinea pigs given, *per os*, infective larvae taken from the earthworm, 50 per cent became infected.—*Vet. Bull, June, 1955.*

Fluorosis in Sheep

Sheep given drinking water containing 0.3 to 20.0 p.p.m. of fluoride showed no adverse effects in health, weight, or wool production. The variation in water consumption resulted in only 4 to 5 mg. of fluorine ingested per day during the winter but 30 to 60 mg. per day during the summer. No mottling or excessive wear of the teeth resulted.—*Vet. Bull., May, 1955.*

Necrosed Round Ligament in Cows

Six cases of postparturient rupture of the round ligament in the hip joint of dairy cows in Tasmania, 5 in 1951 and 1 in 1954, are reported. Two followed prolonged dystocia but in all cases the cow had been unable to rise after calving. In each there were no signs of parturient paresis, the cow would not lie on the affected side, the leg could be abnormally abducted, and crepitation could be auscultated in the affected joint. Necropsy in each case revealed hemorrhage and debris in the joint, with complete degeneration and disappearance of the round ligament in 3, a discolored and degenerated ruptured ligament in 1, and a stretched, flaccid, and easily broken ligament in 2. The condition was bilateral in 1 cow but in the others the opposite round ligament was normal. The cause was undetermined.—*Austral. Vet. J., April, 1955.*

Gamma Globulin for Canine Distemper

Field trials showed that purified distemper antibody in the form of vacuum-desiccated *gamma* globulin had advantages over anti-distemper serum. The globulin minimized the danger of transmitting canine hepatitis virus, contained no preservative, could be stored much longer, was ten times as concentrated, and the cost was comparable to serum. It could also be made bivalent against canine distemper and hepatitis viruses, or polyvalent to include the bacteria which often complicate distemper.—*Vet. Rec., Sept. 18, 1954.*

Rabies in Cattle. II. Review of Immunization of Herbivorous Animals Against Rabies

HILARY KOPROWSKI, M.D.

Pearl River, New York

EACH PROBLEM in the field of rabies investigation presents the challenge, and the ordeal, of singling it out from an intricate maze of similar problems. Only by tracing the history of investigations can one observe the progressive differentiation of apparently similar problems.

Throughout the first decade of the modern era of rabies research, dating from the time of Pasteur, the prevention of human rabies through the treatment of individuals already exposed received most attention. The problem of rabies in domestic animals (and a great deal of attention was paid to rabies in animals by Pasteur[1,8] and his collaborators) was also attacked by attempts to develop a successful method of treatment applicable after exposure to the virus.

Although rabies in dogs was the main object of research, the protective immunization of herbivorous animals was attempted as early as 1881 by Galtier,[2] who proposed the intravenous inoculation of live street virus as a method of prevention. In later experiments, he[3] "vaccinated" sheep intravenously with street virus and later found them "immune" to challenge with the homologous strain administered subcutaneously.

Roux and Nocard[4] introduced 10 to 20 drops of rabbit brain emulsion infected with rabies virus into the veins of 3 cows and 3 calves, but found these animals susceptible to rabies when challenged intraocularly with street virus seven weeks later. When the "immunizing" dose of virus was increased to 3 ml. and administered twice—first, 24 hours following intraocular exposure to street virus; and, second, two days later—3 of 4 sheep survived.

These results were interpreted as indicating that rabies might be prevented in an already exposed (severely) herbivorus animal through the prompt intravenous administration of live rabies virus. Following a similar line of thought, Moncet[5] treated 3 cows bitten by a rabid dog by the intravenous administration of the dog's brain tissue. None of the cows died. The question is, would they have died if they had not been treated at all?

Although some workers[6] have entertained doubts about the safety of the intravenous inoculation of animals with live virus, Krasmitski[7] concluded that slow, intravenous infusion of a virus-infected brain suspension, diluted and preheated at 37 C., was an innocuous method of treatment. He also believed that "vaccination" by the intravenous route resulted in a higher degree of "immunity" than by any other route of administration. The presence of live virus was necessary to secure an immunogenic response.

Turning from street virus to fixed virus, Meiszner[8] attempted to vaccinate calves and sheep with three injections of live virus given at one- to two-day intervals, administered either intravenously or intraperitoneally. In some experiments, animals apparently were found to resist challenge 12 to 20 days after the last injection. However, when intracerebral challenge inoculation (with fixed virus) was given between the second and third immunizing injections, or immediately after the third, the animals succumbed to rabies. Intramuscular challenge with street virus immediately following the series of immunizing inoculations enabled the author to show a high degree of protection of herbivorous animals. The virus used for immunization had to be from freshly harvested rabbit brain tissue, since even the Pasteur method of desiccation made the vaccine ineffective (Schnurer[9]).

Mass immunization of herbivorous animals (but only after assumed or proved exposure to rabies) has been practiced in several European countries since the early twentieth century. In Hungary, during the 25 years beginning with 1902, thousands of cattle, sheep, and swine were "treated" with eight to nine injections of fixed virus, according to the method of Hogyes, which used dilutions of virus for reduction of virulence, rather than drying. Aujeszky,[9] who summarized the results of these immunizations, believed strongly in their effectiveness. For instance, of 228 cattle bitten on the head and subsequently treated, 7.8 per cent died of rabies; whereas, according to the same author, the mortality to be expected in nontreated animals would be 60.0 to 70.0 per cent. Aujeszky went so far as to suggest that antirabies treatment should be administered to herbivorous animals even though several weeks might have elapsed since the actual exposure. This postulate has been challenged by Mello and Poggio[10] who, on the basis of a limited number of experimental observations, advocated the administration of Fermi-type (phenolized) vaccine immediately after exposure to achieve protection. Statistical evalu-

From American Cyanamid Co., Research Division, Lederle Laboratories, Pearl River, N. Y.

*The references for this article appear at the end of this series, on page 369.

ation of antirabies treatment (again *following exposure*) of 851 herbivorous animals vaccinated in the Smolensk province of Russia[11] indicated a death rate of 1.1 per cent, but no comparative figure is given for nontreated animals.

These references are quoted to show that the immunization of herbivorous animals against rabies has been successfully practiced for some 50 years, but as an isolated procedure without direct relation to the problem of the eradication of rabies.

Only in the past two decades has our attention been focused upon a more universal and global approach in the fight against rabies, and the immunization of herbivorous animals must have a place in the development of a comprehensive program. Among the herbivorous animals, cattle seem to be particularly susceptible. This is evidenced not only by high mortality in field exposures to the bites of rabid dogs or foxes, but also by the results of experimental challenge inoculations.[12] In addition, cattle seem to play a dominant role as "blood donors" for hematophagous bats, as well as being "recipients" of rabies from the virus-carrying bats. Until recently, this problem existed only in Central and South America, but it has now been extended by the discovery of rabid bats in the United States.[13,14] Although the latter are not of the blood-sucking variety, it may be postulated that the changed habits of insectivorous bats when rabid may make the transmission of rabies to cattle from bats a serious problem in this country.

Progress in understanding the part played by cattle and other herbivorous animals in the epizootiology of rabies has not been paralleled, unfortunately, by an equally rapid advance in the development and evaluation of prophylactic procedures. This prompted the initiation, some years ago, of the studies reported here. The results represent a cooperative effort of several laboratories, including two in South America, where rabies in cattle often assumes epizootic proportions. Since immunization with the egg-adapted, attenuated, Flury strain of virus has given excellent results in the field of canine prophylaxis,[15] the use of this type of vaccine as a method of immunizing cattle figures prominently in these studies.

Rabies in Cattle. III. Comparative Studies on Vaccination of Cattle in Colombia with Flury Virus and Chloroform-Inactivated Vaccine

CESAR GOMEZ, M.D.; JACK BLACK; HILARY KOPROWSKI, M.D.

Pearl River, New York

The susceptibility of cattle to rabies has been known for many years.[16*] Sporadic attempts at immunization have been made, but studies involving exact procedures have not been reported.[**] Rabies in cattle has been a particular problem in South America[17–19] and, following the successful use of the Flury strain of living rabies virus to immunize laboratory animals and dogs,[20,21] experiments were instituted in Colombia to determine its efficacy in cattle.

MATERIALS AND METHODS

Virus Strains.—The history of the Flury strain of rabies virus has been previously described.[22,23] In the Colombian experiments, virus from the fortieth to fiftieth egg passages was administered to groups of cattle as a 20 per cent suspension of infected chicken embryo, rehydrated from the frozen and desiccated state.

TABLE I—Results of Neutralization Tests on Serums* of Adult Cattle Immunized with Fortieth to Fiftieth Egg-Passage Flury Strain or Kelser Vaccine

Vaccine (ml.)	Route (i.m.)†	Animals vac. (No.)	Age (yr.)	Ratio of tested** serums showing neutralizing antibodies at days after vaccination 0	41	77
Flury(10)	neck	15	2½-8	0/9	9/9	9/9
Flury(20)	neck	15	1¼-7	3/9	9/9	8/9
Flury(20)	thigh	15	1½-4	1/9	8/9	6/9
Kelser(30)	thigh	15	1½-8	0/12	11/12	11/12
Controls	.....	15	2-6	0/14	0/14	0/14

*Undiluted serum; †i.m. = intramuscular injection; **ranch conditions prevented obtaining test serums from all vaccinated animals.

Vaccine containing chloroform-inactivated rabies virus, routinely prepared and distributed by the Instituto Nacional de Higiene Samper-Martinez at Bogota for rabies prophylaxis in cattle, was ad-

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The authors are grateful to Drs. Rafael Gonzales and Alfredo Lleras for their assistance in conducting the field trial.

*The references for this article appear at the end of this series, on page 369.

**The studies reported here actually preceded those of Schroeder et al.,[12] although their work on vampire bat rabies in cattle in Honduras has already been published.

ministered to numerically similar groups of cattle for the purpose of comparing results with those obtained with the live Flury virus. This vaccine (Kelser method) consisted of a 33 per cent suspension of rabies-infected calf brain tissue in physiological saline solution, inactivated with 1 per cent chloroform.

A street strain of rabies virus (NYC), isolated from the submaxillary salivary gland of a dog in New York City, was used for challenge. A 20 per cent suspension of canine submaxillary salivary glands prepared in 10 per cent normal rabbit serum saline was used.

The CVS fixed strain of rabies virus (standard challenge virus supplied by the National Institute of Health to laboratories in the United States) was used in the neutralization tests. Typical range cattle of various breeds, from 1 month to 8 years of age at time of vaccination, were maintained on pasture for a year at a ranch belonging to the Instituto Nacional de Higiene, just outside Bogota. Test cattle were identified by ear tag.

Serum-Neutralization Test.—Equal parts of test serum—undiluted and in serial dilutions of 1:5, 1:25 and 1:125—were mixed with a dilution of guinea pig brain suspension of the CVS rabbit-fixed strain of rabies virus containing 25 to 125 mouse l.d.$_{50}$.† After mixtures were incubated 90 minutes at 37 C., 5 Swiss mice were injected intracerebrally with 0.03-ml. doses and the results recorded as the protective titers of the serums, calculated by the Reed and Muench method.[24]

EXPERIMENTAL

Serological Indications of Immunity in Adult Cattle Following Vaccination.—Seventy-five cattle, 15 months to 8 years of age, were divided into five equal groups. Representative numbers of animals from each group were bled prior to immunization, and serum-neutralization tests were performed. Rabies antibodies were demonstrated in the serums of only 4 animals. Cattle were then immunized intramuscularly as follows: Groups 1 and 2 received 10 ml. and 20 ml., respectively, of the Flury strain in the neck; group 3, 20 ml. in the thigh; and group 4, 30 ml. of the Kelser chloroformized vaccine in the thigh. Group 5 was held as uninoculated controls (table 1).

Serums were again obtained 41 and 77 days after vaccination, with neutralizing antibodies being detected in undiluted samples from 37 of 39 inoculated cattle at 41 days, and from 34 of 39 at 77 days. Serums of control animals showed no neutralizing antibodies at any time.

Serums of some of the vaccinated animals were titrated to determine the degree of antibody response, and the protective titer at 77 days was found to range from 1:4 to 1:140. Neither site of injection nor amount of vaccine appeared to have any bearing on the level of protective antibody.

Immunity in Vaccinated Calves and Young Cattle.—As the above experiment indicated that the Flury strain of rabies virus at the fortieth to fiftieth egg-passage level could be used for immunizing adult cattle under local conditions, and as neutralizing antibodies were present in the blood of animals following vaccination, studies were instituted to measure immune response as indicated by survival after challenge with street virus as well as by the appearance of homologous antibodies. Young cattle, under 2 years of age, were divided into three age groups and were injected with 10 or 20 ml. of the Flury strain vaccine or with 30 ml. of the chloroformized Kelser vaccine (table 2). Calves less than 7 months old were similarly divided into four age groups and received 5 or 10 ml. of Flury or 15 ml. of Kelser vaccine (table 3). All inoculations were made in-

†The dose which will kill 50 per cent of the mice inoculated.

TABLE 2—Results of Serum-Neutralization Tests and Challenge of Young Cattle Immunized Against Rabies with Fortieth to Fiftieth Egg-Passage Flury Strain or Kelser Vaccine

Vaccine	Amount injected* (ml.)	Age group† (mo.)	Animal (No.)	Minimum protective titer of serum, days after vaccination: 0	51	77	365	Results of challenge**	Street virus isolation from salivary gland
Flury	10	6-14	676	<1:2	.....	.....	1:4	Died	Positive
			678	<1:2	1:38	>1:250	>1:250	Survived	.....
			681	>1:2	>1:2	>1:2	.....	Survived	.....
			682	<1:2	1:196	1:66	.....	Survived	.....
Flury	20	20-22	684	<1:2	.....	.....	1:18	Survived	.....
			685	<1:2	.....	.....	>1:250	Survived	.....
			686	<1:2	.....	.....	1:66	Survived	.....
			687	<1:2	1:38	1:66	1:18	Survived	.....
Kelser	30	12-24	688	<1:2	1:85	1:23	1:5	Survived	.....
			689	<1:2	1:18	1:17	1:12	Died	Positive
			690	<1:2	.....	.....	1:91	Survived	.....
			691	<1:2	.....	.....	.....	Died	Positive
			692	<1:2	.....	.....	.....	Died	Positive
None	.....	8-14	693	<1:2	.....	.....	.....	Died	Positive
			694	<1:2	.....	.....	.....	Died	Positive

*Intramuscularly in the thigh. †Five animals were vaccinated in each of the three age groups. Under ranch conditions, it was impossible to obtain test serums from all immunized cattle. **Challenged with NYC strain of rabies virus, 10 per cent suspension of canine salivary gland, 1 ml. into each masseter muscle, on day 365.

tramuscularly into the thigh and one group was left unvaccinated in each experiment. Although 5 animals were vaccinated in each age group, existing field conditions made it impossible to perform both the serological and the challenge tests on all of them.

Serum samples were obtained from certain animals immediately before vaccination, then 51, 77 and, in most cases, 365 days following vaccination. Except in one instance no neutralizing antibodies were detected in the serum samples secured before vaccination. The level of antibodies observed at various intervals following vaccination varied from group to group and differed from animal to animal.

One year after vaccination, all animals, including controls, were challenged with 1 ml. of a 10 per cent suspension of the NYC strain of rabies virus injected bilaterally into each masseter muscle (total 2 ml.) and observations were continued for six months. A satisfactory immune response was demonstrated. All control animals, but only 2 of those vaccinated, died as a result of the challenge (table 2). One which died, the youngest in this group, had received 10 ml. of Flury vaccine; the other, 18 months old, had received 30 ml. of Kelser vaccine. Rabies virus was isolated from the salivary glands of all that died.

In calves less than 7 months of age (table 3), the Flury vaccine seemed to confer better immunity than the inactivated virus. A calf, 1½ months old, immunized with 5 ml. of the Flury strain, survived challenge, as did 5 of 9 slightly older animals injected with 10 ml. of the same virus. All 5 of the Kelser vaccine-immunized (15 ml.) group died, and rabies virus was isolated from the salivary glands of 3. In contrast, no rabies virus was detected in the salivary glands of 3 of the dead Flury-vaccinated calves; the fourth was not tested. Rabies virus was isolated from the brain tissue of these animals, so its absence from the salivary glands is particularly interesting.

COMMENT

The experimental data presented should be considered as an attempt to evaluate antirabies immunization in cattle. When these experiments were conducted (1948), knowledge concerning the potency of the Flury strain vaccine was by no means complete, and the number of animals used was small; therefore, the foregoing data should be considered as preliminary in nature.

However, an analysis indicates that the vaccine which contained living Flury virus showed a good degree of immunizing potency regardless of the size of the inoculum. The neutralization tests seem to indicate as good an antibody response to a 10-ml. as to a 20-ml. dose (the only animal tested in the group which received 5 ml. of Flury vaccine showed neutralizing antibodies 77 days after vaccination).

A 30-ml. dose of Kelser-type vaccine, containing chloroform-inactivated virus, exhibited good antigenic qualities, as indicated by antibody response and the resistance of all but 1 animal to challenge (table 2), whereas none of the animals which received the 15-ml. dose withstood challenge one year later (table 3).

It is worth noting that all animals which

TABLE 3—Results of Serum-Neutralization Tests and Challenge of Calves Immunized Against Rabies with Fortieth to Fiftieth Egg-Passage Flury Strain and with Kelser Vaccine

Vaccine	Amount injected* (ml.)	Age group† (mo.)	Animal (No.)	Minimum protective titer of serum, days after vaccination: 0	51	77	365	Results of challenge**	Street virus isolation from salivary gland
Flury	5	1-1½	902	<1:2	1:96	1:92	.....	Survived	.....
			906	<1:2	>1:250	>1:250	1:28	Survived	.....
			907	<1:2	1:92	1:58	.....	Died	Negative
Flury	10	3-4	908	<1:2	1:96	1:22	1:2	Died	Negative
			909	<1:2	.....	.....	1:22	Survived	.....
			910	<1:2	1:66	1:38	1:8	Survived	.....
			911	<1:2	1:4	1:2	.....	Died	Not tested
			913	<1:2	1:112	1:112	1:167	Survived	.....
Flury	10	4-5	914	<1:2	1:112	1:66	1:10	Survived	.....
			915	<1:2	1:66	1:22	1:22	Died	Negative
			916	<1:2	1:18	1:66	1:10	Died	Positive
			917	<1:2	1:22	1:92	1:18	Died	Positive
Kelser	15	4-7	918	<1:2	.....	.....	1:22	Died	Negative
			919	<1:2	1:12	1:4	1:4	Died	Positive
			920	<1:2	1:5	1:4	.....	Died	Not tested
None	....	5-6	921	<1:2	.....	.....	.....	Died	Positive
			922	<1:2	.....	.....	.....	Died	Positive
			924	<1:2	.....	.....	.....	Died	Positive
			926	<1:2	.....	.....	.....	Died	Positive

*Intramuscularly in the thigh. †Five animals were vaccinated in each of the four age groups. Under ranch conditions, it was impossible to obtain test serums from all immunized cattle. **Challenged with NYC strain of rabies virus, 10 per cent suspension of canine salivary gland, 1 ml. into each masseter muscle.

showed neutralizing antibodies higher than 1:96 dilution survived challenge inoculation. However, the correlation is less obvious in animals with low serum titers. For instance, cow 688 (table 2), vaccinated with 30 ml. of Kelser-type vaccine, survived challenge although its protective serum titer at the time of challenge was only 1:5, while calf 918 (table 3), which received 15 ml. of the same vaccine, succumbed to rabies when its serum showed a protective titer of 1:22. The inconsistencies may have been connected with age differences; however, the number of animals was too small to permit drawing conclusions as to the relationship of the level of circulating antibodies and resistance to infection.

While the street virus was isolated from the salivary glands of only 1 of 4 animals, which died after vaccination with the living Flury virus, it was found in the salivary glands of 4 of 5 animals which died after inoculation with Kelser-type vaccine. While this has relatively little importance, from the epizootiological point of view, since rabid cattle rarely, if ever, transmit rabies by biting, it is of importance in several other species.

SUMMARY

Field studies, in Colombia, on the immunization of cattle against rabies indicated that good protection was induced with the Flury strain of living virus, and with a large (30-ml.) dosage of chloroform-inactivated vaccine, whereas a smaller dose (15 ml.) of the latter seemed to have no immunizing power in this experiment.

Rabies in Cattle. IV. Vaccination of Cattle with High Egg-Passage, Chicken Embryo-Adapted Rabies Virus

HILARY KOPROWSKI, M.D.; JACK BLACK; WILLIAM P. JOHNSON, D.V.M.

Pearl River, New York

The control of rabies in dogs is of critical importance in preventing the spread of the disease to man, whereas rabies in cattle is chiefly an economic problem for the farmers and ranchers. It is causing considerable concern in all cattle-producing countries. The report of the Animal Disease and Parasite Research Branch, U.S. Department of Agriculture, states that, of 8,837 cases of animal rabies in 1953, 1,012 occurred in cattle.[25*] The estimated loss from rabies in cattle in the spring of 1952 in only four counties in Georgia was $100,000.[26]

Some cattle have been vaccinated with phenolized or ultraviolet-irradiated vaccines, but reports are scattered and insufficient to assess the efficacy of the procedure. In view of successful results in the immunization of laboratory animals and dogs with the Flury strain of modified live rabies virus, studies were undertaken to determine the effectiveness of this vaccine in the immunization of cattle.

The history[23] of the Flury strain will be only briefly reviewed here. The strain was isolated by Leach and Johnson[21] from tissues of a girl who had died of rabies. Johnson (cited in[23]) carried the virus through 136 intracerebral passages in day-old chicks. In this laboratory, after two additional chick-brain passages, the virus was adapted to the developing chicken embryo. Koprowski and Cox[22] reported reduced virulence of the Flury strain at fortieth to fiftieth egg-passage levels for hamsters and guinea pigs, and loss of virulence for rabbits injected parenterally. In later studies by Koprowski and Black,[23] dogs inoculated parenterally with virus which represented levels between the twentieth and eightieth egg passages were found to resist challenge by inoculation with a virulent street strain of rabies virus. Vaccine prepared from the fortieth to fiftieth egg-passage level of the Flury strain was then mass field-tested, and since April, 1950, more than 1,700,000 dogs all over the world have been vaccinated with this agent.

Cattle rabies has been a problem in South and Central America for some years. The previous report in this series (Part III, p. 360) recounts studies on the immunization of cattle in Colombia with the fortieth to fiftieth egg-passage Flury vaccine. Later, Schroeder and his associates[17] carried out experiments in Honduras, Costa Rica, and Guatemala in which 6,087 calves and adult cattle were vaccinated with the same vaccine. One to five dog doses (5 to 15 ml.) were administered by various routes. Rabies was not reported in any vaccinated cattle, although the prevalence of the disease remained high in nonvaccinated cattle in the same areas.

Subsequent field use of the low egg-passage Flury vaccine in one section of New York State, however, indicated a lack of uniformity in the safety of the vaccine for cattle. Of 639 cattle vaccinated with material representing the fortieth egg passage, 2 died and 8 manifested incoordination and hindleg weakness. The 8 animals recovered, but 1 remained partially paralyzed.

From American Cyanamid Co., Research Division, Lederle Laboratories, Pearl River, N.Y.

*The references for this article appear at the end of this series, on page 369.

Meanwhile, in the laboratory the serial passage of the Flury strain in chicken embryos was continued[19] and another change in the properties of the virus was noted when the series reached the 176th and 182nd egg passages. The virus became apathogenic for adult mice, rabbits, and dogs when injected intracerebrally. Adult mice and hamsters which survived intracerebral inoculation with suspensions of the high egg-passage (HEP) virus were found to be immune upon challenge with street virus.[20] In addition, the intramuscular inoculation of dogs, cattle, and primates with this HEP virus induced a state of resistance to challenge with street virus.[20] This indicated that the HEP Flury virus might be used effectively for immunization and might have, at the same time, an added margin of safety over the low egg-passage vaccine for certain species, particularly cattle.

Laboratory studies were, therefore, undertaken to evaluate the HEP virus as an immunizing agent in cattle, and to determine the correlation between serological evidence of immunity as measured by neutralization tests and protection as demonstrated by survival following challenge. Also reported here are the results of a field trial in Florida to determine the effect of various dosages of the HEP virus on the antibody response in cattle of various ages.

LABORATORY STUDIES

MATERIALS AND METHODS

Virus Strains.—Live Flury-strain rabies virus from above the 175th egg passage was used in all vaccination experiments. A 33 per cent suspension of infected chicken embryo was filtered through two layers of gauze and was then desiccated from the frozen state. It was rehydrated to the same value at time of use.

Challenge was performed by the bilateral intramasseter injection of 1 cc. of the NYC strain of rabies virus[20] in dilutions made from a 20 per cent suspension of canine submaxillary salivary glands in 85 per cent normal rabbit serum-glycerin kept frozen at -60 C.

Animals.—Cattle were bought at random from various farms. The calves were between 3 and 6 months, the cattle between 6 and 12 years, of age at the time of vaccination. Swiss albino mice weighing 10 to 12 Gm. were used for virus-titration purposes and in serum-neutralization tests.

Serum-Neutralization Test.—The serum-neutralization technique consisted of mixing equal parts of undiluted test serum and a dilution of guinea pig brain suspension infected with CVS rabbit-fixed strain of rabies virus and containing 40 to 100 $L.d._{50}$. After the mixtures had been incubated for 90 minutes at 37 C., 0.03-ml. doses were injected intracerebrally into mice. Deaths of mice occurring between six and 21 days were considered due to rabies. The final $p.d._{50}$* was calculated by the method of Reed and Muench.[24]

Laboratory Examination.—Rabies as the cause of death in cattle was determined by microscopic examination of brain tissue smears for Negri bodies, and by the inoculation of mice with brain and salivary gland tissue.

EXPERIMENTAL

Immunizing Properties of High Egg-Passage Flury Virus.—Two groups of 5 calves each were inoculated intramuscularly in the thigh with one injection of 10 and 15 ml. of the HEP Flury strain, respectively (table 4). Thirty days later, the 10 vaccinated calves and 5 unvaccinated controls were challenged by the inoculation of 1 ml. of a 1:100 dilution of the NYC virus bilaterally into the masseter muscles (2 ml. total) and all were observed for approximately one year. Of the 5 unvaccinated controls, 4 died of rabies 29, 32, 33, and 46 days after challenge, as did 2 of the calves which had received the 15-ml. dose of vaccine, 21 and 22 days after vaccination. All calves in the group receiving 10 ml. survived challenge.

TABLE 4—Immunizing Properties of Different Doses of HEP Flury Strain of Rabies Virus Injected Intramuscularly in Calves

	Vaccination*		Challenge†	
			Mortality ratio	
Dose (ml.)	Date	No and age of animals	Date	Controls D/T**	Vaccinated D/T
15	8/19/52	5 (3-6 mo.)	9/19/52	4/5	2/5
10	8/19/52	5 (3-6 mo.)	9/19/52	4/5	0/5

*There were no postvaccination reactions; †31 days postvaccination with rabies street virus; **D/T = number of deaths per total animals.

Correlation Between Resistance to Challenge and Serological Evidence of Immunity.—In another experiment (table 5), groups of calves were inoculated intramuscularly in the thigh with one injection each of 3, 6, 10 or 15 ml. of the HEP Flury strain. Thirty days after vaccination, blood samples were obtained for serum-neutralization tests. At the same time, the NYC strain of virus was prepared in dilutions of 1:30, 1:90, and 1:270, and the calves in the various groups, together with unvaccinated controls, were challenged by injection.

TABLE 5—Correlation Between Resistance and Serological Evidence of Immunity Induced in Calves by HEP Flury Strain of Rabies Virus

Dilutions of challenge virus	Ratio of calves: (A) showing serum-neutralizing antibodies, and (B) surviving challenge with street virus, after immunization* with different amounts of Flury strain									
	3 ml.		6 ml.		10 ml.		15 ml.		None †	
	A	B	A	B	A	B	A	B	A	B
1:30	3/3	3/3	3/3	3/3	2/3	3/3	3/3	3/3	0/2	0/2
1:90	3/3	2/3	2/4	2/4	3/3	3/3	3/4	3/4	0/3	0/3
1:270	2/2	2/2	2/3	2/3	3/3	3/3	2/3	2/3	1/3	0/3

*Intramuscular route; ** unvaccinated control animals.

With the exception of 1 calf given 3 ml. of vaccine and challenged with 1:90 dilution of

*The dose which will protect 50 per cent of the animals inoculated ($p.d._{50}$).

TABLE 6—Serum-Antibody Response of Cattle 30 Days After Injection of HEP Flury Strain of Rabies Virus

Vaccine			Antibody response/number of cattle injected
Egg passage	Route of injection	Amount (ml.)	
185th	Masseter (i.m.)	1.5	4/10*
		3.0	6/10
	Thigh (i.m.)	1.5	10/10
		3.0	8/10
		6.0	6/9
	Triceps (i.m.)	6.0	7/10
187th	Neck region (i.d.)	1.0	11/11

*The denominator indicates the number of cattle bled; the numerator, the number of animals whose serum showed the presence of antibodies; i.m. = intramuscular; i.d. = intradermal.

virus, animals whose serum showed neutralizing antibodies survived challenge which killed all controls. The data indicate that as little as 3.0 ml. of the HEP Flury rabies virus produced a satisfactory immune response, and that there was close correlation, under the conditions of the test, between the protection evidenced by survival following challenge and the presence of antibodies in the blood. Since the smallest dose of virus seemed to induce as good protection as larger dosages, it may be assumed that the bovine host became immune because the Flury virus multiplied in its body.

The Effect of Route and Dosage on Protection Afforded.—Seventy adult cattle were divided into seven equal groups and serum samples were subjected to neutralization tests (table 6). With one exception, antibodies against rabies were entirely lacking. The cattle were then vaccinated with one injection of HEP Flury virus as follows: 1.5 or 3.0 ml. into the masseter muscle; 1.5, 3.0 or 6.0 ml. into the thigh muscles; 6.0 ml. into the triceps brachii; and 1.0 ml. intradermally in the neck. From the practical point of view, the masseter route of inoculation proved to be the least satisfactory because of the difficulty in restraining the animals and variation in muscle thickness.

Thirty days after vaccination, serum samples were again secured and tested for antibodies against rabies. Considering the over-all response with various dosage levels used, inoculation into the thigh, intramuscularly, and in the neck, intradermally, produced a better immune response than injection into either the triceps brachii or masseter muscles. Although there were differences among the ratios of animals developing antibodies after the inoculation of different dosages of vaccine into the thigh, these variations may have been attributable to experimental error.

FIELD TRIAL IN FLORIDA

Since laboratory studies indicated the safety and effectiveness of the HEP Flury strain of rabies virus in cattle and antibody protection was demonstrated by serum-neutralization tests, a carefully supervised field trial was planned. The experiments were planned to investigate the effect, as shown by antibody response, of different amounts of the HEP Flury vaccine given in one or two injections to cattle of various ages.

MATERIALS AND METHODS

The Flury strain of virus from above the 175th egg passage was used for vaccination. Undiluted and serial fivefold dilutions of serum were subjected to neutralization tests in mice against 5 to 90 $l.d._{50}$ of fixed virus. More detail is given under the section on "laboratory studies."

Animals.—Forty Brahman cross calves, 3 to 15 months of age, and 40 cattle 2 to 15 years of age at the time of vaccination, were identified by ear tag and tattoo.

TABLE 7—Comparative Response in Antibody Formation in Cattle and Calves Vaccinated with Either One or Two Injections* of HEP Flury Strain of Rabies Virus

				Vaccination Schedule				
				1 Injection		2 Injections		No. of animals with increased protective titer after 2nd injection
	Dose of vaccine	No. of animals vaccinated		Ratio of tested† animals with neutralizing antibodies‖ days after vaccination				
Animal	(ml.)	1 Injection	2 Injections	30	60	30**	60‡	
Adults	3.0	10		7/9	6/8			
			10			8/9	9/9	6
	1.5	10		3/9	8/10			
			10			7/9	9/9	7
Calves	3.0	10		4/9	6/10			
			10			10/10	10/10	7
	1.5	10		5/9	7/9			
			10			7/10	9/10	7

*Intramuscularly into the thigh. †Under field conditions, it was impossible to run tests on all animals originally vaccinated. ‖The lowest antibody titer considered protective was 1:5 against five $l.d._{50}$ doses of virus. **30 days after second injection.

EXPERIMENTAL

The animals were vaccinated in groups of 10 with one or two injections of 3.0 ml. or 1.5 ml. of a 33 per cent suspension of the HEP Flury virus intramuscularly in the thigh (table 7). Thirty days later, serum samples were obtained for neutralization tests and half the animals were revaccinated with a dose of HEP virus equal to that which each had originally received. Animals were again bled 30 days later (60 days after the first inoculation) and the serums subjected to neutralization tests.

The lowest antibody titer considered positive was 1:5 against five $l.d._{50}$ doses of virus. Thirty days after the first vaccination, the figures indicate that the 3.0-ml. inoculum induced a better response than did the 1.5-ml. inoculum. Fifteen of 18 adult cattle and 14 of 19 calves which had received one or two inoculations with the larger amount possessed antibodies, compared with 10 of 18 adults and 12 of 19 calves for the smaller dosage. When the serums of animals which had received only one injection were tested 60 days after vaccination, the two amounts of vaccine appeared to induce immunity in comparable numbers of animals. There was a definite increase in the number of both cattle and calves in the 1.5-ml. dosage groups which showed protective titers 60 days after one inoculation of vaccine as compared with the 30-day results.

In almost all cases, the second injection improved the immunogenic response. All of the adult cattle, all calves which were inoculated with 3.0 ml., and all but 1 of the calves inoculated with 1.5 ml. of vaccine possessed antibodies against rabies in significant amounts 30 days following the second injection. The last column in table 7 gives the number of cattle in which the second injection produced a "booster" effect, that is, in which the serum antibody titers were increased.

COMMENT

Data presented in the section of this article dealing with laboratory investigations indicate that the HEP Flury strain produces an immunogenic response in cattle injected intramuscularly. In the preliminary experiments (table 4) challenge inoculation with street virus lethal to 4 of 5 controls killed only 2 of 10 vaccinated animals. The results of the second experimental series were also encouraging, and an almost perfect correlation was obtained between the serological and challenge data. It was also of interest that homologous antibodies and resistance to challenge were induced by as small an amount as 3 ml. of vaccine, a dose equivalent to that used for the immunization of dogs.

Data obtained in the field trial in Florida confirmed the fact that a small (1.5 ml.) amount of HEP virus may immunize cattle, and induce formation of antibodies in the majority of the inoculated animals although in some cases, particularly in adult cattle, antibody response seemed to be delayed until 30 to 60 days after inoculation. In those animals which received one dose of 3 ml. of vaccine, "improvement" of the antibody status on the sixtieth day was observed in only 2 of 18 animals. Revaccination of cattle 30 days following the first immunization resulted in a general "booster" effect. Five of 6 animals which failed to develop antibodies 30 days after the first inoculation of vaccine responded with the formation of antibodies after the second inoculation. In the remaining animals, the level of circulating antibodies rose following the second inoculation of vaccine.

In addition to these studies, a field study was initiated in Georgia, where 1,107 cattle of various ages, from nursing calves to adults, were inoculated with one dose of 15 cc. of the HEP virus.[26] Over a six-month observation period, no reactions were observed and there were no deaths from rabies. Six and one half months after vaccination, representative numbers of vaccinated cattle were challenged. Solid immunity was demonstrated in 11 of 16 (69%) to challenge which resulted in death of 12 of 16 (75%) unvaccinated controls. Thus, the results of field observations, though limited, seem to confirm the data obtained in the laboratory indicative of the immunogenic power of the HEP Flury strain in cattle.

Rabies in Cattle. V. Immunization of Cattle in Brazil Against Exposure to Street Virus of Vampire Bat Origin

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Paralytic rabies in cattle and horses has been reported in epizootic proportions in Brazil since 1908. In 1911, Carini[16] observed Negri bodies and isolated rabies virus from the brain tissue of cattle found dead during an epizootic in the state of Santa Catarina, in southern Brazil. The

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mass destruction of dogs in the vicinity seemed to have no effect upon the incidence of the disease. In fact, rabies in dogs was rarely found in the affected areas, and serious consideration was given to a belief existing among farmers of the region that the disease was caused by the bites of vampire bats. Carini quoted reports that vampire bats flying in broad daylight—an activity abnormal for this species—had been seen to attack cattle which subsequently died. From 1916 onward, extensive epidemiological studies were conducted in southern Brazil by Haupt and Rehaag.[20] They assembled important data indicating the role of the vampire bat in rabies transmission, and they isolated rabies virus from a captured bat. However, the definite demonstration of rabies transmission by these vectors came much later as a result of studies conducted in Trinidad by Hurst and Pawan[19,21,22] and in Brazil by Queiroz Lima and Torres.[33,34]

Control of epizootics of rabies in Brazil has been accomplished by the vaccination of livestock in the affected areas. The vaccine has generally been fixed virus employed in the form of a phenolized emulsion of cattle or horse brain tissue. Since the production of these vaccines is laborious and expensive, an attempt was made to evaluate the protection of cattle by immunization with the living Flury strain of rabies virus against exposure to a vampire bat strain. Results obtained by Schroeder *et al.*[12] in Honduras, and by Koprowski *et al.* (part IV, p. 363) in laboratory exposures, were encouraging enough to justify the present investigations.

MATERIALS AND METHODS

Virus Strains.—The HEP* of the Flury strain of rabies virus has been described in earlier publications.[20] A 33 per cent suspension of 188th egg-passage infected chicken embryo, stored in the desiccated state, was used for immunization.

The Itatiba strain of rabies virus was employed for challenge. This strain was originally isolated from the brain of a horse which died of rabies in Itatiba, São Paulo, Brazil, after exposure to the bite of a vampire bat. The virus was then passed through two guinea pig passages, and a brain tissue suspension from the second passage was injected into 3 dogs and 2 calves. All of these animals died, and virus was isolated from their brains. A suspension of infected dog brain tissue was next injected into guinea pigs. Brain tissue

*High egg passage.

TABLE 8—Antibody Response and Results of Challenge in Brazilian Cattle Vaccinated with High Egg-Passage Flury Virus

Vaccine (ml.)	Animal (No.)	Minimum protective titer of serum 30 days postvaccination*	Challenge† results**	Death days after challenge	Isolation of virus	Mortality ratio
	11	1:4	Died	11-20 days	Yes	
	12	1:13	Died	151	No	
	13	1:5	Died		Yes	
	14	1:4	Died		Yes	
1.5	15	1:4	Died	11-20	Yes	10/10
	16	<1:2	Died	days	Yes	
	17	1:4	Died		Yes	
	18	1:8	Died		Yes	
	19	<1:2	Died		Yes	
	20	>1:5	Died	150	No	
	21	1:22	Survived	———	———	
	22	1:13	Survived	———	———	
	23	1:76	Died	110	No	
	24	1:13	Died	123	No	
5.0	25	1:4	Survived	———	———	4/10
	26	>1:190	Survived	———	———	
	27	1:17	Survived	———	———	
	28	1:13	Died	139	No	
	29	1:76	Survived	———	———	
	30	1:18	Died	149	No	
	31	1:87	Survived	———	———	
	32	1:87	Survived	———	———	
	33	1:3	Not challenged	———	———	
	34	<1:2	Not challenged	———	———	
6.0	35	1:4	Died	143	No	1/6
	36	1:18	Survived	———	———	
	37	1:18	Survived	———	———	
	38	<1:2	Survived	———	———	
	40	1:6	Not challenged	———	———	
Unvaccinated Controls	1-10	———	Died	11-20 days	Yes	10/10

*Against 70 $l.d._{50}$ fixed virus; **six months after challenge; †challenged 48 days after vaccination.

of the latter was used for challenge in this present study.

Animals.—Grade yearling cattle were used throughout the experiments. They were housed, and identified by ear tags.

EXPERIMENTAL

Antibody Response in Animals Injected with the Flury Strain.—Forty yearlings were divided into four groups of 10 each. All animals were bled immediately before vaccination, and none of the serum samples indicated a significant level of antibodies against rabies. Three groups were inoculated intramuscularly in the thigh with one injection of 1.5, 3.0, or 6.0 ml., respectively, of the HEP Flury strain, and the fourth group was held as unvaccinated controls (table 8). Thirty days after vaccination, the animals were bled again and the serums subjected to neutralization tests. Antibody titers were low in the group which received 1.5 ml. of vaccine. They were appreciable, with no material difference, in the groups given 3.0- and 6.0-ml. doses of vaccine.

Titration of the Challenge Virus in Unvaccinated Animals.—Before the Itatiba virus was employed for challenge of vaccinated cattle, an attempt was made to measure its virulence for unvaccinated cattle. A dilution of 1:10, which had infected all of 4 cattle inoculated in previous tests, was chosen for challenge purposes.

Serial twofold dilutions, ranging from 1:20 to 1:320, of the infected guinea pig brain suspension were made. Seven cattle were injected with each of the five dilutions (table 9). Six weeks after inoculation of the virus, the mortality ratios among the different groups were identical. Surprisingly enough, at the end of an 18-week observation period, the mortality ratios had changed. Additional animals had died but the changes affected all dilutions regardless of the amount of virus present; thus, the titration gave no basis for determining the exact $l.d._{50}$ of the virus in the challenge material. However, it was clear that the dilution used for challenge represented an unusually high concentration of virus.

TABLE 9—Results of Titration* of the Itatiba Strain of Rabies Virus in Normal, Unvaccinated Cattle

Dilution of virus	Mortality ratio	
	6 weeks postinoculation	18 weeks postinoculation
1:20	4/7	6/7
1:40	4/7	5/7
1:80	4/7	5/7
1:160	4/7	5/7
1:320	4/7	5/7

*The virus, 1 ml., was injected into the masseter muscles (2 ml. total).

Results of Challenge Inoculation of Vaccinated Cattle.—Forty-eight days after immunization, both vaccinated animals and unvaccinated controls were challenged by the inoculation into each masseter muscle of 1 ml. of a 1:10 dilution of the guinea pig brain suspension infected with the Itatiba virus.

The results of challenge (table 8) must be interpreted in relation to the time elapsing between challenge and the recording of results. By the twentieth day after challenge, all 10 control animals and 8 of 10 vaccinated with 1.5 ml. of Flury virus were dead, and rabies virus was isolated from the brain tissue of each by subinoculation into mice, but no animal was either sick or dead in the two groups which received 3.0 or 6.0 ml. of the vaccine. At this point, a perfect correlation existed between the results of serological examination and resistance to challenge, since the surviving animals in the group given 1.5 ml., cows 12 and 20, possessed a seemingly higher level of neutralizing antibodies than those which succumbed to rabies.

The challenge results remained the same during the next three months, and then 7 more animals died: 1 in the group given 6.0 ml. of vaccine, 4 in the group given 3.0 ml., and the 2 survivors in the group given 1.5 ml. No virus was isolated when brain-tissue suspensions of these animals were injected intracerebrally into mice, making it impossible to assess the significance of these late deaths. These results are the more puzzling because of the apparent lack of correlation between the antibody level and the death of the animal. For instance, cow 23, which had an antibody level of 1:76, 30 days postvaccination and 18 days before challenge, died 110 days after challenge; whereas cow 25, with an antibody level of 1:4, survived. Also, animals 27 and 30 had almost identical antibody levels, yet 1 died 149 days after challenge and the other survived. It is possible, of course, that antibodies may have disappeared from the blood of the animals which died in the course of the four to five months between antibody determination and death. The "disappearance" of the virus from the brains of the animals presents another puzzle. Although the possibility of "self-sterilizing neuroinfection"[88] may be considered as the explanation of the phenomenon, the presence of virus in brains of animals which died earlier does not support such a theory.

Thus, whatever caused the death of the 7 last animals, one must interpret the results in the light of the experiment as a whole. It is obvious (table 8) that the animals were subjected to a severe challenge test, 8 of 10 of those inoculated with 1.5 ml. of vaccine having succumbed within three weeks. The serological response of those inoculated with 3.0 ml. was good, as judged by the level of antibodies observed 30 days after vaccination and by the response to challenge. Increasing the dose of vaccine to 6.0 ml. seemed to elicit even a better immune response.

The results, in general, indicate that cattle vaccinated with the live Flury strain virus not only may become immunized against rabies street virus

of canine origin (NYC strain) but also against a South American strain of vampire bat origin.

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EDITORIAL

Disease Control by Insanitation

An authority has said that breeding of animals for improvement of stock, including breeding for resistance to disease, is net gain whereas other means of improving production are a continuous operation expense. This theory seemingly is being given a field test by certain currently popular practices in the control of poultry diseases.

The theory may be interpreted to mean that true disease control is most effectively accomplished by breeding for resistance—while disease prevention and the treatment of disease are only stop-gap measures. Still, many cling to the concept that improvement of environment or prevention of disease by every means, including nutrition, is worthy of comprehensive study and application. To indicate how preventive medicine has benefited mankind they point to the decreasing infant mortality and to man's longer life expectancy. Likewise, they point out that the eradication of the Texas fever tick so improved the environment that cattle could be raised safely and that such an environment is now maintained at a very small "operating expense." They emphasize the same for foot-and-mouth disease and for fowlpest. They credit the near eradication of bovine tuberculosis with so improving the environment of man that children no longer suffer the agonizing effects of tuberculosis of the bones or of the pulmonary or central nervous systems and other types of tuberculosis caused by exposure to infected cows; likewise, the eradication of glanders was of benefit to mankind.

On the other hand, it is quite obvious that present medical measures save the lives of some individuals, both man and animal, that could not survive rugged nature and thus their relatively inferior germ plasms remain in the species. Since no one is willing to judge which person should live and which might better not live, for the benefit of posterity, man must continue to study measures for the alleviation, control, and eradication of his diseases. Similar studies continue on the diseases of his livestock, ultimately for the welfare of man, a point apparently not appreciated by those who shortsightedly would discontinue all research on domestic animals. At the same time, there are those who believe in animal disease suppression through breeding almost to the exclusion of environmental means of control.

Poultry Research Shows the Way

The advancement of knowledge of the infectious diseases of the larger domestic animals has been slow while that of nutrition and of the infectious diseases of poultry has been comparatively rapid. Because the funds available were not sufficient for comprehensive studies on larger animals, the available facilities were rightly directed toward diseases of poultry. This has caused some question as to whether the experiment stations have the personnel to undertake research on the diseases of larger animals. The magnificent job these stations have done on poultry diseases should provide the answer.

The writer well remembers when the poultry of the Middlewest was not affected with the many diseases now ubiquitous; when pullorum disease existed only in a few flocks, usually where unsuspecting poultrymen, in their efforts toward "improvement" of their flocks, had imported the disease with chickens from pullorum-disease areas. Unfortunately, advertised "superior breeding stock" not uncommonly are carriers of disease.

With every advance there usually are certain bad features. With the advent of the commercial hatchery, numerous poultry diseases spread like wildfire. Pullorum disease was one of the first maladies so disseminated. It was soon followed by leukosis. Too late it was discovered that chickens free of leukosis could not be obtained with certainty. Essentially all flocks were infected or became infected. There was nothing that could be employed as an "immunizing" agent, which may have been fortunate because materials employed for

This editorial was written by Dr. S. H. McNutt at the request of the editor.

the production of immunity are sometimes without merit, regardless of the scientific literature. Some of the oldest vaccines on which the public has come to depend are without truly proved merit. Nor was there any "cure" for leukosis.

Resistance by Breeding

An early worker (the late F. D. Patterson) on leukosis was among the first to advocate production and reproduction from known infected flocks, to gradually build up strains that were resistant to leukosis. In this attempt, hatching flocks have been culled repeatedly to eliminate the individuals with signs of leukosis, leaving the birds that were most apt to be resistant. For this purpose, 2-year-old birds were obviously more desirable than younger ones, yet some were carriers and spreaders. There is ample evidence that this procedure has been effective to a degree. However, with leukosis still a major killer of chickens, it is evident that it is not adequate.

Resistance by Exposure

Essentially the same situation can be shown for most poultry diseases. However, with coccidiosis it is slightly different. All chickens raised under "practical" conditions are expected to be exposed to coccidiosis, usually at an early age. The objective, therefore, is to allow controlled exposure, through the use of deep litter, while protecting the birds with anticoccidial drugs with the expectation that active immunity will develop. The goal is a subclinical or inapparent infection. The partial success in the control of coccidiosis by this method has made popular the transfer of "old" or "used" litter to new or clean poultry houses—in environments previously free of poultry diseases. Here, also, the objective is to expose the birds to a low level of common infection in the hope that they will acquire resistance. What is this if not planned insanitation?

We are elated with the great advances in knowledge of fowlpox, Newcastle disease, bronchitis, laryngotracheitis, chronic respiratory disease, and others, but when we consider the application of this knowledge we must be disappointed. Once more it is proposed that poultry be exposed to these diseases—truly "control of disease by insanitation."

It is well known that unlimited use of living vaccines, even some so-called "avirulent" agents, has established disease on premises and that often, when the so-called "vaccination program" has been initiated, it must be continued year after year. The use of living vaccines has been of benefit under certain circumstances but the advisability of the over-all use of such vaccines in all domestic birds may be questioned. One wonders if the damage resulting from such procedure has not largely offset the benefits.

It must be concluded that intentional or forced exposure to disease, which to make it more respectable is called vaccination or even immunization, has kept dollars in the pockets of many poultrymen. It must follow that such "vaccination" has put dollars in the pockets of others. Is it not possible that dollars speak louder than logic?

Thus, there is promoted the idea that poultry, especially chickens, must be exposed to coccidiosis, to the diseases (both known and unknown) found in "used litter," to leukosis, Newcastle disease, chronic respiratory disease, bronchitis, laryngotracheitis and pox—all before they reach laying age. Often such forced contact with disease is multiple. Currently, it is recommended that any bird kept beyond the broiler age be exposed at least three times to Newcastle disease vaccines. It is a veritable miracle that so many chickens do withstand such violent and repeated assaults on their health and well-being. Since many chickens can not withstand such an environment, the question is raised as to whether we can add still more infections to those which chickens must survive—whether we have not already gone too far.

While the control of disease by "insanitation" has, as yet, been applied extensively only to poultry, it is increasingly evident that the same result is obtained, often unknowingly, in the diseases of larger animals, especially the enteric diseases of calves and pigs. To evaluate the influence of such environments, comparisons must be made with "normal" animals—an uncertain term unless a considerable amount of disease is considered "normal." Fortunately, methods of obtaining standard (disease-free) pigs have been developed for research. We are making progress.

However, for the present we must welcome disease prevention, control, and eradication by every means possible and as rapidly as those means can be developed.—S. H. McNutt, Madison, Wis.

CURRENT LITERATURE

ABSTRACTS

Arthritis in Swine

The administration of 75 mg. of cortisone subcutaneously daily for four weeks, 50 mg. daily for one week, and 25 mg. daily for one week produced marked clinical improvement in 3 advanced chronic cases of arthritis due to *Ery. rhusiopathiae* infections. A marked reduction in the erythrocyte sedimentation rate was produced by ACTH in 4 arthritic animals but the rates rose to the original level or higher three weeks after withdrawal of hormone therapy. These animals had pathological changes in the kidneys and adrenals. In arthritic swine with severe renal and adrenal damage, ACTH may be contraindicated, since adrenal rest, not stimulation, is needed.—[*Dennis Sikes, George M. Neber, and L. P. Doyle: Studies on Arthritis in Swine. II. The Effects of Hormonal Therapy on Advanced Chronic Polyarthritis Experimentally Induced by Erysipelothrix Infections. Am. J. Vet. Res., 16, (July, 1955): 367-373.*]

Infections with Mixed Etiology

A plea is made for more thorough and critical study of factors influencing the development of disease in domesticated animals. Emphasis is placed on Koch's postulates and particularly the experimental production of a disease. Production under controlled conditions is not impossible since the very existence of a disease is evidence that it can be done. Inability to produce, experimentally, a disease as it occurs in nature means a lack of understanding of all the factors involved. No original or new data are given. The concept of two or more etiological factors operating in concert to produce a disease is illustrated by examples from the literature. These examples include a concerted action of a virus and a bacteria, two different bacteria, a virus and a chemical compound, a virus and a parasite, bacteria and alteration of enzyme systems of the host, two different viral agents, and a virus plus a high environmental temperature.—[*Carl Olson, Jr.: Infections with Mixed Etiology. Am. J. Vet. Res., 16, (July, 1955): 458-462.*]

Antibiotic Blood Levels in Cows

From this study with cattle, in comparison with aqueous suspensions, it is believed that: (1) benzathine in sesame oil with 2 per cent aluminum monostearate produces much lower penicillin blood levels during the first 24 hours. After 24 hours, the levels are identical in duration with those produced by the aqueous suspension, but the penicillin concentration may be slightly higher. (2) benzathine and procaine penicillin G in sesame oil wth 2 per cent aluminum monostearate produce somewhat lower penicillin blood levels during the first 24 hours, after which it may be slightly higher with an effective duration of six days, similar to that produced by the aqueous suspension. (3) streptomycin sulfate administered in sesame oil with 2 per cent aluminum monostearate produces streptomycin blood concentrations of greater duration than do aqueous suspensions.—[*C. J. Hollister, R. A. Huebner, William B. Boucher, and Thomas DeMott: Bovine Antibiotic Blood Levels Obtained with Antibiotics in Oil Suspensions. Am. J. Vet. Res., 16, (July, 1955): 391-393.*]

Distemper in Wild Carnivores

Thirty-five feral carnivores including 14 gray foxes (*Urocyon cinereoargenteus*), 3 red foxes (*Vulpes fulva*), 16 raccoons (*Procyon lotor*), and 2 skunks (*Mephitis mephitis*), were presented with rabies-like symptoms such as aggressiveness, depraved appetite, and frequenting populated areas. Histological and mouse-injection tests eliminated the likelihood of rabies. Canine distemper was diagnosed in 20 animals based on either the presence of pathognomonic inclusion bodies or ferret inoculations. Of the remaining 15, some were believed to have canine distemper based on (1) similarity of clinical history to those of proved cases; (2) the exclusion of rabies; and (3) the observation that distemper inclusion bodies were not always present in feral animals with distemper, confirmed by ferret inoculation. Encephalitis was seen in 20 animals, not always associated with canine distemper. Other notable findings were 1 raccoon infected with *Eurytrema procyonis*, and 2 with *Dracunculus insignis*. Urinary capillariasis was common in all species.—[*C. F. Helmboldt and E. L. Jungherr: Distemper Complex in Wild Carnivores Simulating Rabies. Am. J. Vet. Res., 16, (July, 1955): 463-469.*]

Differentiation Between Foot-and-Mouth Disease and Vesicular Stomatitis Viruses

The paper reviews previous work with the viruses of foot-and-mouth disease and vesicular stomatitis in suckling and adult white mice. The expense and practical problems of using cattle, horses, and other large animals to check negative results obtained with the complement-fixation test are also discussed. Experiments were conducted with two strains of each of the three classical types of foot-and-mouth disease virus and the two types of vesicular stomatitis virus. Each strain of virus was inoculated intraperitoneally into suck-

ling mice (7 to 8 days old) and intracerebrally into adult mice (30 to 50 days old). Foot-and-mouth disease virus proved to be infective for suckling mice but not for adults while vesicular stomatitis virus appeared to be considerably more pathogenic for adults than for suckling mice by these routes of injection. Complement-fixation tests on tissues from the dead mice revealed that, with foot-and-mouth disease virus, suckling mouse muscle gave consistently positive results, while brain tissue was negative. With vesicular stomatitis virus, brain tissue antigens from both age groups gave positive fixation; muscle antigens from adult mice were consistently negative but on one occasion a suckling mouse muscle antigen gave strong fixation. On the basis of these results a simple and inexpensive routine test has been devised to check material received for examination which gives negative results with the complement-fixation test.—[*Raymundo G. Cunha, Ervin A. Eichhorn, and Fidel Mata O.: Differentiation Between Foot-and-Mouth Disease and Vesicular Stomatitis Viruses by Means of Mouse Inoculation. Am. J. Vet. Res., 16, (July, 1955): 472-480.*]

FOREIGN ABSTRACTS

Fluorine Poisoning in Cattle

Chronic fluorine poisoning in cattle was found to be caused by a high percentage of fluorine in the grass and water which were contaminated with acid fluorine aerosols. The symptoms were manifested by malformation of the developing enamel of the teeth; formed enamel was not damaged. It is not advisable to raise young animals on contaminated areas. There was a significant difference in the urine fluorine content of animals which received sodium and those which received sodium fluoride plus tenfold doses of aluminum sulfate. It is advisable to give aluminum sulfate in the form of specially prepared cakes to animals which ingest fluorine.—[*J. Tesink: Fluorine Poisoning in Cattle. Tijdschr. voor Diergeneesk., March 15, 1955.*]—L.V.E.

The Technique of Bovine Fetotomy

An inquiry to 30 veterinary practitioners about the use of the fetotome in cows revealed that 60 per cent prefer to have the cow standing, as it is easier; 40 per cent prefer the cow to be lying, as it is safer; and 80 per cent prefer a complete fetotomy to a partial dismemberment and traction which frequently mutilates the dam. The following were considered important: a good epidural anesthesia; having the cow's hindquarters elevated; having a lying cow in a completely lateral position; planning the entire fetotomy before beginning; having a wire saw of good quality and an experienced layman to handle it; contacting the calf with the fingers during the entire operation; limiting all movements into and out of the birth canal to a minimum; and postoperative antibiotic therapy.

An uncomplicated fetotomy should take about 15 minutes but with an oversized calf, an abnormally narrow tract, or an emphysematous fetus, a few hours may be required. About 2 per cent of bovine dystocia cases can not be solved by fetotomy. The number of cases requiring fetotomy varies with the breed and with the practice area. Some veterinarians do 25, others up to 120 fetotomies per year. Dystocia is so common in certain strains or breeds of cattle that this should be considered in selecting sires for artificial insemination.—[*M. van de Plasse. The Technique of Bovine Fetotomy. Vlaams Diergeneesk. Tijdschr., Feb., 1955.*]—L.V.E.

Copper Deficiency in Cattle

In four dairy herds, when a diagnosis of copper deficiency seemed justified this diagnosis proved to be correct in only one. In the other three herds, the low serum-copper level and the symptoms could be explained by the insufficient care of the pasture, parasitic infections, and other causes. In two herds, copper was administered without benefit. Mistakes are made when all possible causes of the symptoms are not taken into account.—[*W. A. Eisma, E. G. Hoskum, W. Dorsman, and G. Waeringer: Observations Regarding the So-Called Deficiency of Copper for Bovines. Tijdschr. voor Diergeneesk., March 15, 1955.*]—L.V.E.

Bovine Abortion from Salmonellosis

The authors described a case of abortion at the sixth month of gestation in a cow from which *Salmonella dublin* had been isolated.—[*J. Deom and J. Mortelmans: Bovine Abortion from Salmonellosis. Tijdschr. voor Diergeneesk., Nov., 1954.*]—L.V.E.

Concretion in the Stomach of a Horse

In the abattoir of Breda, a concretion was found attached to the stomach of a horse and completely enclosed. The weight of the concretion was 246 Gm. (8 oz.). It was grayish yellow, with a rough surface, and was formed in layers. Internally, there were several cavities in which a small amount of gray substance was observed. The chemical composition was about 80 per cent calcium carbonate.—[*P. H. Van Diessen and A. J. A. Berkemeyer. A Stomach Stone in a Horse. Tijdschr. voor Diergeneesk., Sept. 15, 1954.*]—L.V.E.

Hormonal Castration

The author concludes that a hormonal castration which suppresses the function of the germinative glands and the development of secondary sexual characteristics until now has been reasonably certain only in cocks. This was achieved with synthetic estrogen substances. Under the influence of estrogen substances, the ovary of the hen remains uninfluenced, but the ovary of quadruped mammals inclines to luteinization and anestrus. The function of the testicle of mammals is not effectively

retarded by the dosages and methods used. The favorable effects on growth and the quality of the meat which are observed in chickens, to some extent in sheep and cattle, and sometimes in pigs must be compared with the results of castration.—[*M. Tausk: About Hormonal Castration. Tijdschr. voor Diergeneesk., Jan. 1, 1955.*]—L.V.E.

Listeria Monocytogenes in the Netherlands

A serological examination of 24 strains of *Listeria monocytogenes* isolated in the Netherlands revealed a division in two groups. Thirteen strains belonged to type I Patterson (structure I, II, III AB): one strain was from a Silver fox; two from man; four from chickens; one from a cow; three from bovine fetuses; and two from pigs. Eleven strains belonged to type IV Patterson (structure III, V, AB, C); one from man; two from chickens; two from cattle; one from a bovine fetus; three from pigs; one from sheep; and one from a canary. Type II and III Patterson were not found.—[*J. Donker-Voet: Serological Examinations of a Few Strains of Listeria Monocytogenes Isolated in the Netherlands. Tijdschr. voor Diergeneesk., Sept. 15, 1954.*]—L.V.E.

Pseudolyssa in Belgium

The author submits a description of the first cases of pseudolyssa (pseudorabies) in Belgium.—[*A. van Wassenhove: Pseudolyssa in Belgium. Vlaams Diergeneesk. Tijdschr., Feb., 1955.*]—L.V.E.

BOOKS AND REPORTS

Glasser's Disease

A fibroid inflammation of the serous membranes and fibroid arthritis of young pigs, previously regarded as acute hog plague (pasteurellosis) appears in all parts of Germany and in many foreign countries. Young pigs are the only animals affected. The author, in 1910, showed that bipolar bacteria were not the cause of the malady. The disease usually appears a few days after transportation or a change of environment or feed. The pigs lose their appetite, develop a fever and a variable number of arthritic symptoms and, sometimes, a painful cough. Some show nervous symptoms and convulsions, then a rapidly spreading paralysis. They may die in two to five days. Perhaps only the youngest of the litter is affected.

Pathological changes include albuminous, watery, gray to yellowish liquid in the peritoneal and pleural cavities, the heart sac, and sometimes in the subdural spaces and cerebral ventricles. A grayish yellow, dry, fibrous skin (film) develops on the serous membranes and the synovial membranes become hyperemic. Joints may be completely filled with a fibrous mass. To a lesser degree the liver, kidneys, heart, and small portions on the spleen become swollen.

The disease always follows stress factors such as moving, cold, trauma, prolonged hunger, unhygienic conditions, and changes to extremely rich rations. Bacteria are found in the inflammatory exudates in most cases, usually pure cultures of a rod bacillus, but sometimes several shorter bacteria resembling the bipolar bacterium are also present. The organisms apparently perish quickly since none can be found in some cases. Mayerhoffer found the rod bacillus in 40 per cent. Hiarre and Wramby, in 160 cases, usually found this bacterium in pure culture. They agreed that it perishes and disappears. They transmitted the disease with pure culture but not regularly and only with unnatural methods—intraperitoneally, subdurally, intravenously, and usually after long transportion. Because they believed the bacterium identical with the influenza bacterium of Pfeiffer, Schope, and Kobe, they recommended that the disease be called "influenza". However, "grippe" is a contagious respiratory disease.

The bacterial infection alone is not the cause of the disease. The disease must be differentiated from pasteurellosis, viscosum infection, streptococcosis (all by bacteriological means), and from pyogenic infections, usually following wound (castration) infections with development of a grayish green exudate in the peritoneal and perhaps the pleural cavities.

Prophylactically, stresses such as overfeeding and exposure to cold during transportation should be avoided with young pigs. As a treatment, salicylic acid preparations have been replaced by sulfonamides.—[*Glasser, Hupka, and Wetzel: Die Krankheiten des Schweines. 5th ed. M. and H. Schaper, Hanover, Germany (1950): 106-112.*]—FRANK A. TODD.

Soil

This book is intended to give the student of agriculture an insight into the new science of soil management. It contains chapters on the architecture and the chemistry of soil, the fauna and the flora of the soil, humus, soil classification, soil erosion, etc.—[*Soil. By G. V. Jacks. 221 pages. 10 illustrations. Philosophical Library, Inc., 15 E. 40th St., New York 16, N. Y. 1954. Price $5.00.*]—W.A.A.

Microbiology

This is a textbook for the student of general and pathogenic microbiology. Since the first edition was published in 1949, there have been many new developments in immunology, disease agents, antibiotics, and other features. The first part emphasizes phylogenetic groups and the biology, physiology, and control of the microbial population; the second part treats of parasitism and disease, immunity, the unicellular pathogens, community health, and preventive microbiology. The relationship of pathogenic microorganisms to other microorganisms, as well as the host itself, is emphasized.—[*Microbiology. By Florence C. Kelly and K. Eileen Hite. 2nd ed. 615 pages. 207 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, N. Y. 1955. Price $7.50.*]—W. A. AITKEN.

THE NEWS

Dr. Casorso—Ralston Purina Fellowship Winner

Dr. Donald R. Casorso, Storrs, Conn., who was awarded the 1955-1956 Ralston Purina fellowship in veterinary science, received his M.S. degree in poultry nutrition and management from the University of British Columbia, and his D.V.M. from Ontario Veterinary College in 1955. His graduate work will be taken at the University of Connecticut, Storrs. His anticipated project will be to establish the normal histology of the avian respiratory tract and then, having established what is normal, study the pathological lesions resultant from the more common respiratory diseases. He is also interested in establishing a rapid diagnostic test for respiratory diseases.

s/H. A. GRAFF, *Educational Division, Ralston Purina Company.*

Joint Southern and Florida V.M.A. Meeting

The joint meeting of the Southern V.M.A. and the Florida V.M.A. in Jacksonville, Fla., Oct. 16-19, 1955, promises to be one of the largest and best ever held in the Southeast. Fourteen guest speakers from various parts of the country will take part in the program. In addition to the combined meeting, the southern section of the American Animal Hospital Association will also be in session.

The meetings will be held in the George Washington Hotel in Jacksonville. All veterinarians are cordially invited to attend. Dr. A. A. Husman, Raleigh, N. Car., is secretary of the Southern association and Dr. Robert P. Knowles, Miami, is secretary of the Florida V.M.A.

s/STANLEY C. WASMAN, *Resident Secretary, Florida.*

Eastern Floods and Veterinarians

At the time of going to press, the AVMA central office had received no reports of loss of life, extensive property damage, or other injury to veterinarians in the eastern states most severely affected by floods following "Hurricane Diane."

Drs. S. F. Scheidy, Philadelphia, Pa., and Edwin Laitinen, West Hartford, Conn., Executive Board members in Districts II and IX, respectively, were asked to survey their areas and report. Although hundreds of private citizens and small businesses suffered great losses, it appears that veterinarians generally in the flood zones escaped serious damage to buildings, equipment, and supplies.

The rapid organization of relief by the Red Cross, plus federal aid and assistance from all over the country, have done and will do much to assuage the shock of the flood disaster.

The AVMA and the JOURNAL will appreciate further reports from veterinary associations and members.

WOMEN'S AUXILIARY

New Officers of Auxiliary.—At the thirty-eighth annual meeting of the Women's Auxiliary to the AVMA, held during the annual meeting of the AVMA in Minneapolis, Aug. 15-18, 1955, the following officers were elected: Mrs. Earl N. Moore, Wooster, Ohio, president; Mrs. Alfred E. Coombs, Skowhegan, Maine, president-elect; Mrs. Lewis H. Moe, Stillwater, Okla., first vice-president; Mrs. E. A. Woelffer, Oconomowoc, Wis., second vice-president; Mrs. U. E. Marney, San Antonio, Texas, third vice-president; Mrs. Frank R. Booth, Elkhart, Ind., secretary; Mrs. C. M. Rodgers, Blandinsville, Ill., membership secretary; Mrs. John D. Stevens, Sequim, Wash., treasurer; Mrs. E. E. Leasure, Manhattan, Kan., recorder; and Mrs.

Mrs. Earl N. Moore, Wooster, Ohio, is the new president of the Women's Auxiliary.

(Continued on page 377)

News From Washington

Unless something unforeseen develops, the Air Force does not expect to issue orders calling additional **veterinary reserve officers to active duty prior to April, 1956.** Those to be called during the current calendar year have received, or soon will, orders for active duty. It was expected that most of the 1955 veterinary graduates who had accepted reserve appointments would be called for duty during the calendar year; however, budgetary and other factors changed the picture. The Army situation is somewhat similar, except that each Army headquarters has been notified to issue orders covering Department of Army requirements for veterinary reserve officers for each calendar quarter to include June 30, 1956.

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Dr. Howard W. Johnson was appointed **acting chief,** Animal Disease and Parasite Research Branch, Agricultural Research Service, U.S.D.A., effective Sept. 1, 1955.

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As a result of an **outbreak of equine encephalomyelitis in certain New England** states the latter part of August, the chief of the Inspection and Quarantine branch, ARS, U.S.D.A., based on a request from Canadian veterinary officials, directed all field veterinarians to see that horses offered for entry into Canada must be accompanied by a certificate issued or endorsed by the U.S.D.A. veterinarian in charge, or his assistant. The certificate must show that the horses have been inspected, the premises of origin, and that the horses were found free of evidence of contagious and infectious disease or exposure thereto, in so far as could be determined. The eastern strain of equine encephalomyelitis virus was recovered from brain tissue of cases submitted to the Montgomery, Ala., virus laboratory from a state in the area affected. It is reported the disease is an extremely virulent type in that the affected animals either die or are in such condition after two or three days that they have to be destroyed.

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Since July 1, **scrapie was diagnosed in one flock of sheep in Iowa, and in six flocks in Indiana.** All sheep in the Iowa outbreak, exposed and infected, were slaughtered; two of the Indiana flocks were slaughtered, and the other four were being slaughtered Sept. 6, 1955.

★ ★ ★

The **major changes provided in the House-passed amendments** (HR-7225) **to the Social Security Act** are: extension of coverage to all self-employed professional persons, except doctors of medicine; reduction of retirement age for women from 65 to 62 years; payment of disability insurance benefits to permanently and totally disabled persons at age 50, if certain other eligibility requirements are met (no benefits given dependents of such workers); continuation of benefit payments to permanently and totally disabled children after age 18, provided the disability existed prior to reaching age 18, and increases in the tax rate paid into the social security program by covered individuals. The tax rates, current, ultimate, and proposed, on self-employed persons are:

Effective dates	Present schedule pays	*Proposed schedule pays
Jan. 1, 1955	3 %	3¾%
1960	3¾%	4½%
1965	4½%	5¼%
1970	5¼%	6 %
1975	6 %	6¼%

*The tax rate applies to the first $4,200 of self-employed net earnings, tax being paid to the Bureau of Internal Revenue with the income tax.

HR-7225 passed the House of Representatives, July 18, 1955, but was not acted on by the Senate prior to adjournment of the First Session, 84th Congress. The Senate Finance Committee is expected to hold public hearings early in the next session.

(Continued from page 375)

L. R. Richardson, Ravenna, Ohio, retiring president. The following appointments were also made: for editor of the *Auxiliary News*, Mrs. H. S. Atkins, Flint, Mich.; historian, Mrs. V. H. Miller, Charleston, W. Va.; parliamentarian, Mrs. B. S. Pomeroy, St. Paul, Minn. The nominating committee for the coming year is composed of Mrs. J. A. Muffly, chairman, Lewisburg, Pa.; Mrs. Harry Hodges, Ithaca, N. Y.; and Mrs. R. C. Klussendorf, Terre Haute, Ind.

Further information and pictures of convention activities will appear in the November JOURNAL.

s/(MRS. FRANK R.) LEONORE M. BOOTH, *Secretary.*

STUDENT CHAPTER ACTIVITIES

For copy deadline, see "Among the States and Provinces"

Michigan Chapter.—The following officers will serve the University of Michigan Student Chapter of the AVMA during the fall semester: Ernest A. Liptak, president; Atwood Asbury, vice-president; Aaron Leach, treasurer; and Ellin Abbott, secretary. The faculty advisors are Drs. John P. Newman and Mark Rines.

s/ERNEST A. LIPTAK, *President.*

APPLICATIONS

Applicants — Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

BICKLEY, C. C.
Box 591, Billings, Mont.
D.V.M., Kansas State College, 1950.

BROWN, WILLIAM W., JR.
117 W. Lake St., Fort Collins, Colo.
D.V.M., Colorado A. & M. College, 1944.

BURGESS, JOHN J.
204 E. Third St., Wabasha, Minn.
D.V.M., Cornell University, 1929.

ENGLE, DAVID F.
2111 Water St., Wesleyville, Pa.
D.V.M., Kansas State College, 1932.

HOWELL, NORMAN H.
971 St. Anthony Ave., St. Paul, Minn.
D.V.M., Kansas State College, 1927.

IKARD, ELIJAH H.
322 Colorado, Gooding, Idaho.
D.V.M., Kansas State College, 1919.

MOORE, WILLIAM M.
Metter, Ga.
D.V.M., Alabama Polytechnic Institute, 1943.

MOSBACH, L. E.
307 3rd St., N., Humboldt, Iowa.
D.V.M., Iowa State College, 1930.

O'MARA, WILLIAM D.
1557 S. Bellevue, Memphis, Tenn.
D.V.M., Texas A. & M. College, 1949.

PEACOCK, GERALD V.
2503 San Pablo Ave., Berkeley, Calif.
D.V.M., Iowa State College, 1950.

RANKIN, LLOYD G.
Forestville, Wis.
D.V.M., Michigan State University, 1941.

TUFTS, FERGUS A.
Western Ave., Morristown, N. J.
M.R.C.V.S., Veterinary College of Ireland, 1944.

WEBSTER, CHARLES, JR.
711 N. Virginia, Roswell, N. M.
D.V.M., Colorado A. & M. College, 1950.

YOUNG, VARLEY F.
P. O. Box 1297, Natchitoches, La.
D.V.M., Texas A. & M. College, 1950.

Applicants — Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

First Listing

HUBBEN, KLAUS
522 Penn St., Newtown, Pa.
V.M.D., University of Pennsylvania, 1953.
Vouchers: M. W. Allam and W. E. LaGrange.

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25 Elder Ave., Yeadon, Pa.
D.V.M., Colorado A. & M. College, 1940.
Vouchers: J. G. Hardenbergh and F. Cross.

QUIGLEY, JOSEPH S.
Quartermaster Inspection Service Command, Santee St., 5th Floor, Los Angeles, Calif.
D.V.M., Iowa State College, 1943.
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REED, IRVIN T.
Area Veterinarian, 6003 SU, Fort Ord, Calif.
D.V.M., Colorado A. & M. College, 1932.
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D.V.M., Texas A. & M. College, 1954.
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D.V.M., Texas A. & M. College, College Station, Texas, 1944.
Vouchers: T. Ramos and B. B. Alfredson.

Second Listing

CURSACK, HORACIO A., Moreno 2001, Esperanza, Prov. de Santa Fe, Argentina.
LOPEZ-SECO, JORGE A., Trelles, 1040, Buenos Aires, Argentina.
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TOPACIO, TEODULO M., JR., T-163 B, Area 2, University of the Philippines, Quezon City, Philippines.
ZAMBRAMO, ALVARO L., Calle 40, #7-39 Bogota, Colombia, S. A.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

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Cornell University*

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CHING, CLARENCE H. Y., D.V.M., 2264 Kanealii Ave., Honolulu, Hawaii.
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JOHNSON, KEITH T., D.V.M., 1114 Second St., Knoxville, Iowa.
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KEMPERS, GARY J., D.V.M., 119 4th Ave., N.E., Sioux Center, Iowa.
KLEAVELAND, JAY C., D.V.M., Sioux Rapids, Iowa.
LEVAN, ROBERT H., D.V.M., Box 113, New Richmond, Wis.
LOEHRL, ALFRED A., D.V.M., Cambridge, Wis.
LOWRY, WILLIAM E., D.V.M., Rt. 1, Liberty, Mo.
LYNCH, PAUL J., D.V.M., Riverside, Iowa.
MCGEHEE, EUGENE H., JR., D.V.M., 627 Seventh St., Ames, Iowa.
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MOODY, RICHARD A., D.V.M., 182 N. 4th St., Old Town, Maine.
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AMONG THE STATES AND PROVINCES

The deadline for news copy is the 24th of the month, two months preceding the month of issue

Arizona

Central Association.—The regular monthly meeting of the Central Arizona Veterinary Medical Association was held at Chandler on August 9. Dr. Roy Echeverria, Casa Grande, was host. The program consisted of an open discussion and agreement on a set of minimum fees recommended for large animal practice. The discussion was led by **Drs. Jack Fuller,** Phoenix; **Donald Watt,** Glendale; and **John Carney,** Chandler.

s/KEITH T. MADDY, *Secretary.*

Canada

Dr. Gwatkin Retires.—Dr. Ronald Gwatkin (ONT '19), of the Animal Disease Research Institute at Hull, Que., retired in June, after 36 years of eminent veterinary service as a teacher and research worker. He is author or co-author of over 150 scientific papers, chiefly related to the diseases of poultry and swine.

• • •

Maritime Associations.—The three Maritime Veterinary Associations (New Brunswick, Nova Scotia, and Prince Edward Island) held their sixth annual joint conference at Mount Allison University, Sackville, N. B., on June 28-30, 1955, with 55 veterinarians in attendance.

The meeting was opened by **Dr. Chas. A. Mitchell,** chief of the Animal Pathology Division, and the program consisted of the following: greetings from the president of the Canadian Veterinary Medical Association, **Dr. R. McG. Archibald,** Truro, N.S.; major and minor elements in plant and animal growth, **Dr. George R. Smith,** director, Chemistry, Soils and Fertilizer Services, Truro, N.S.; experiences in swine practice, **Dr. R. J. Pinkney,** Cooksville, Ont.; surgical problems in cattle, **Dr. F. J. Milne,** Department of Medicine and Surgery, Ontario Veterinary College, Guelph; canine treatment for the busy "mixed" practitioner, and feline therapeutics, **Dr. Claude Kealey,** Ottawa, Ont.; developments in artificial insemination, **Dr. D. G. Moore,** Provincial Veterinary Branch, Fredericton, N.B.; biological warfare, **Dr. Chas. A. Mitchell,** Hull, Que.; large animal forum, **Dr. W. M. Mutrie,** moderator, Woodstock, N.B.; and scrapie, **Dr. P. J. G. Plummer,** Animal Diseases Research Institute, Hull, Que.

At the banquet, attended by 94 veterinarians and their wives, the **Rev. R. G. Rowcliffe,** prison psychologist at nearby Dorchester Penitentiary, spoke on the problems of penitentiary inmates. The banquet was followed by a program of entertainment with **Dr. G. E. Myers** of Middleton, N.S., as master of ceremonies.

s/J. F. FRANK, *Resident Secretary.*

District of Columbia

Dr. A. W. Miller Dies.—Dr. Arthur W. Miller, former chief of the Bureau of Animal Industry, U.S.D.A., died September 1, at the age of 79, after an illness of several years.

Dr. Miller was born in Manchester, N. H.,

Dr. A. W. Miller

in 1876, and graduated from the Kansas City Veterinary College in 1901. He immediately entered the BAI and served continuously until his retirement in October, 1945. He assisted in making the diagnosis of foot-and-mouth disease in Michigan in 1914 and was in charge of the quarantine of the Chicago stockyards during that epizootic. He served as assistant chief of the Bureau under Dr. John R. Mohler for 16 years, then as chief from 1943 to 1945.

He is survived by a daughter and three sons, one of whom, Donald (OSU '36), is stationed at Phoenix, Ariz., where he is in charge of the Cooperative Livestock Disease Eradication Projects for the Agricultural Research Service, U.S.D.A.

Georgia

Personal.—Dr. James Lieberman, veterinary training consultant with the training branch of the Communicable Disease Center of the U. S. Public Health Service in Atlanta, has recently returned from a two-month assignment in Geneva, Switzerland, where he served as veterinary public health consultant to the European Regional Office of the World Health Organization of the United Nations.

Indiana

Tenth District Association.—The monthly meeting of the Tenth District (Ind.) Veterinary Medical Association was held at Richmond on July 21. The veterinarians of Liberty were hosts to the group. After the business session, veterinarians and their wives enjoyed dancing.

Officers of this Association are Drs. F. A. Gossett, Greenfield, president; R. S. Ensign, New Castle, vice-president; and W. E. Sharp, Union City, secretary-treasurer.

s/J. L. KIXMILLER, *Resident Secretary.*

• • •

Northwestern Association.—On July 28, members of the Northwestern Indiana Veterinary Medical Asssociation enjoyed a picnic in Columbian Park in Lafayette where swimming and other activities were available.

s/J. L. KIXMILLER, *Resident Secretary.*

• • •

Ninth District.—The Ninth District (Ind.) Veterinary Medical Association held a social meeting in the Abe Martin Lodge of the Brown County State Park near Nashville, Ind., on August 19. No business session was planned and members enjoyed swimming and the beauties of the park.

s/J. L. KIXMILLER, *Resident Secretary.*

Louisiana

State Association.—The fourth annual fall meeting of the Louisiana Veterinary Medical Association, Inc., was held August 24-25 at the Washington Youree Hotel in Shreveport.

The program included the following speakers and their subjects: **Drs. C. Edwin Hofmann,** Tulsa, Okla., short cuts in small animal practice; **W. O. Greene,** Nashville, Tenn., free-hand ear trimming; **R. J. Beamer,** School of Veterinary Medicine, Texas A. & M. College, College Station, general surgery in small animals; **W. J. Gibbons,** School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, mucosal disease of cattle, and sterility in cattle; **E. E. Saulmon,** Agricultural Research Service, Baton Rouge, brucellosis eradication in Louisiana; **John R. Dick,** Fort Dodge Laboratories, Fort Dodge, Iowa, diseases of swine; **W. O. Greene,** Nashville, Tenn., horse practice. **Dr. T. W. Leonard,** Bastrop, was toastmaster at the banquet.

s/R. B. LANK, *Secretary.*

Michigan

Dr. Frank Thorp, Jr. (1SC '26), professor of veterinary pathology at the state university for many years, died September 9, while on vacation in Colorado. Details will be in the November JOURNAL.

Missouri

Kansas City Association.—At the July 19 meeting of the Kansas City Veterinary Medical Association in the Livestock Exchange Building, **Dean A. H. Groth,** School of Veterinary Medicine, University of Missouri, was moderator of a panel of clinicians from the University. They presented current phases of their work and discussed recent experimental developments.

The August meeting of the Association was the annual picnic at Dr. J. Knappenberger's farm near Olathe, Kan.

s/BUSCH MEREDITH, *Secretary.*

North Carolina

Officers of State Association.—The following officers have been elected to serve the North Carolina Veterinary Medical Association for the coming year: Drs. B. D. Dawsey, Gastonia, president; R. L. Williams, Raleigh, president-elect; D. C. Beard, Concord, vice-president; C. J. Lange, Greensboro, secretary-treasurer; and C. W. Young, Mocksville, and W. D. Collins, Winston-Salem, to the executive committee.

s/MARTIN P. HINES, *Resident Secretary.*

Pennsylvania

Keystone Association.—The newly elected officers of the Keystone Veterinary Medical Association are Drs. Richard Huebner, Havertown, president; Charles Raker, Philadelphia, vice-president; Jack Emas, Lansdowne, recording secretary; Raymond C. Snyder, Upper Darby, corresponding secretary; and Joseph F. Skelly, Philadelphia, treasurer.

Members voted to continue their veterinary

emergency service for the coming year. This insures prompt professional care for animals in the Philadelphia area at all hours. Anyone unable to reach his regular veterinarian, may call the emergency number and contact the veterinarian on duty in a particular locality.

s/RAYMOND C. SNYDER, *Recording Secretary.*

Virginia

State Association.—The semiannual meeting of the Virginia Veterinary Medical Association was held at the Hotel Roanoke in Roanoke on July 17-19, 1955.

Dr. S. G. Eddins, president of the Virginia Veterinary Medical Association.

The program speakers and their subjects were: the **Hon. Parke C. Brinkley,** commissioner of agriculture, Commonwealth of Virginia, veterinarian-livestock industry relationship; **Drs. W. L. Bendix** and **M. E. Hibbard,** Richmond, veterinarian's role in civil defense; **A. F. Raney,** ARS, Washington, D. C., tuberculosis eradication; **B. V. Favata,** Rochester, N.Y., diseases and injuries of the nervous system; **R. W. Engel,** Blacksburg, animal nutrition; **W. J. Gibbons,** Alabama Polytechnic Institute, Auburn, Ala., diseases of the genital organs of bulls; **R. G. Schirmer,** Michigan State University, East Lansing, small animal practice; **R. J. Byrne,** Grayson Foundation, College Park, Md., equine virus abortion; and **V. B. Robinson,** Zionsville, Ind., malignancies in animals.

A social hour and dinner and dance were enjoyed by the members, their wives, and guests. The program of the women's auxiliary featured a coffee, luncheon, flower arrangement demonstration, and card party.

s/WILSON B. BELL, *Secretary.*

STATE BOARD EXAMINATIONS

Texas—The Texas State Board of Veterinary Medical Examiners announces that the next veterinary licensing examination will be held Jan. 11-13, 1956, at Austin, Texas. The completed applications must be returned to the following address not later than 30 days before the examination date. Requests for applications and additional information should be addressed to: Mr. T. D. Weaver, Executive Secretary, Texas State Board of Veterinary Medical Examiners, 520 Littlefield Bldg., Austin 15, Texas.

VETERINARY MILITARY SERVICE

Short Course in Pathology of Diseases of Laboratory Animals.—A short course in the pathology of diseases of laboratory animals is scheduled for Dec. 5-9, 1955, inclusive, at the Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington, D.C. This is the second year this course has been offered; however, it is expected that none of the subject matter will be repeated.

The course is designed to provide training for professional officers who have charge of procurement and maintenance of animal colonies and is intended particularly to help them interpret natural diseases which may influence the supply of laboratory animals or their suitability for experimental use. It is aimed especially at the needs of various Army, Navy, and Air Force laboratories, most of which have veterinary officers in charge of their animal colonies. Pathology will be the theme of the course, but this facet will be used as a point of departure for discussion of etiology, diagnosis, and control of the diseases under consideration.

The course will be of value only to those individuals qualified to understand disease processes and to absorb information in the field of pathology. Veterinary pathologists should find the course of particular benefit, but pathologists, veterinarians, and others with similar professional backgrounds will also find the course of value.

For information as to the eligibility requirements, qualifications, application procedures and selection of students, refer to Circular 621-9, Department of the Army, Washington 25, D. C., dated June 4, 1955. Civilians should apply to: Director, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, 6825 16th St., N.W., Washington 25, D. C.

s/CAPTAIN W. M. SILLIPHANT, *Deputy Director, Medical Corps, U. S. Navy.*

Historical Meeting in the Far East

Army and Air Force veterinary officers assembled June 23-24, 1955, at the Washington Heights Club in Tokyo for the first military veterinary medical conference ever held under United States auspices in the Orient. It is planned to hold such conferences annually as is done by the veterinary officers stationed in Europe.

First row (left to right)—Major Wm. A. Bridenstine, Major Wm. D. McMonagle, Major Warren J. Schneider, Lt. Col. Russell M. Madison (Air Force), Lt. Col. Howard C. Maxey, Col. Ben F. Leach (Air Force), Dr. Kogi Saito, Col. Neil O. Wilson, Lt. Col. Hugh B. Campbell, Lt. Col. Ralph O. Anslow, Lt. Col. Wesley A. Clem, Jr., Major Francis L. Thomas.

Second row—First Lt. Edwin H. Wung (Air Force), First Lt. M. Dale Dahlquist (Air Force), First Lt. Charles D. Dake (Air Force), Capt. William J. Welch (Air Force), Major Wm. Ginn, Major Paul A. Rainey, Capt. John Q. Adams, Jr., Major Bruce S. Ott, Major Elmer R. Pede, First Lt. Wm. H. Bunting, Jr., First Lt. Frederick J. Burke (Air Force), First Lt. Dale H. Parker, Jr. (Air Force).

Third row—Capt. Wallace L. Anthony, First Lt. N. A. Luckeroth (Air Force), First Lt. E. J. Morelli (Air Force), Major George E. Ritter, Major Lorenz L. Beuschel, Major Wm. J. Morley, First Lt. Harty S. Powell, First Lt. Klaus Hubben (Air Force), First Lt. James N. Brogger (Air Force), Capt. John D. McCullough (Air Force), Capt. B. U. Lauderdale (Air Force).

The fourth Class in the veterinary radiological health course at Oak Ridge, Tenn. This group of Army and Air Force veterinary officers (mainly Army, Air Force is indicated) completed the course given at the Oak Ridge Institute of Nuclear Studies, Inc., from May 31 to June 11, 1955.

Front row (left to right)—Lt. Col. Bernard F. Trum, Oak Ridge; Col. Don L. Deane, Fort Riley, Kan.; Col. Walter Smit, Veterinary Food Inspection Service, Chicago; Col. Thomas Ward, area veterinarian, Seattle, Wash.; Dr. Ralph T. Overman, chairman of the Special Training Division, Oak Ridge Institute; Col. Alpheus H. Seeley, QM Inspection Service Command, Philadelphia; Col. Robert A. Boyce, Fort Bragg, N. Car.; and Major Max M. Nold (Air Force), Oak Ridge.

Second row—Major Joseph S. Quigley, QM Inspection Service, Los Angeles, Calif.; Lt. Col. Nels F. Christensen, First Army Veterinary Inspection Unit, New York City; Lt. Col. George D. Batcheldor, Fort Snelling, Minn.; Capt. Floris M. Garner, Fort Douglas, Utah; Major Robert O. Linder, QM Inspection Service Command, Richmond, Va.; Lt. Col. Mulford C. Lockwood, QM Subsistence School, Chicago; Lt. Col. Wiley H. Horn, QM Depot, Richmond, Va.; and Capt. Thomas G. Murname, Medical Field Service School, Fort Sam Houston, Texas.

Back row—Major Leslie E. Meckstroth, Sixth Army, San Francisco, Calif.; Major James Armstrong (Air Force), Camp Dietrich, Md.; Capt. James E. Cook (Air Force), Armed Forces Institute of Pathology, Washington, D. C.; Lt. Col. Horace R. Collins, General Depot, Memphis, Tenn.; Lt. Col. Clinton R. Gould, Veterinary Food Inspection Service, Chicago; Lt. Col. Arthur Lee Hogge, Fort Sam Houston, Texas; and Lt. Col. Conley G. Isenberg, Fort Sill, Okla.

BIRTHS

Dr. (KSC '53) and Mrs. John H. Hurlburt, Leonardville, Kan., announce the birth of their second child, a daughter, Kathleen Jane, on May 20, 1955.

Dr. (COL '54) and Mrs. Dee Taylor, Ontario, Ore., announce the birth of twin sons, Sullivan and Christopher, on June 28, 1955.

Dr. (TEX '47) and Mrs. Ben B. McCollum, Jr., Stephenville, Texas, announce the birth of a son, Ben Martin, on July 23, 1955.

Dr. (OKL '53) and Mrs. Louis E. Carlin, Collinsville, Okla., announce the birth of a daughter, Susan Louise, on July 26, 1955.

Lieutenant (COL '54) and Mrs. Jack N. Sohrbeck, Fort Totten, N. Y., announce the birth of a daughter, Deborah Lynn, on Aug. 14, 1955.

DEATHS

Carl W. Babcock (ONT '16), 68, Plymouth, Ohio, died April 8, 1955. Dr. Babcock, a general practitioner, was active in civic affairs.

Clarence J. Ballowe (IND '13), Oklahoma City, Okla., died May 23, 1953. Dr. Ballowe had been employed by the U. S. Bureau of Animal Industry.

Edgar H. Callander (ONT '91), 84, Zanesville, Ohio, died June 29, 1955. Dr. Callander had practiced in Zanesville for 63 years. He is survived by his widow; three sons, one of whom is Dr. (MCK '17) W. G. Callander of Parkersburg, W. Va.; five grandchildren and nine great-grandchildren.

Tracy J. Coulter (ONT '21), 61, Cambridge Springs, Pa., died July 6, 1955. Dr. Coulter had also practiced in Los Angeles, Calif., and Erie, Pa. He is survived by his widow and a daughter.

Charles E. Dille (CVC '04), Cairo, Ill., died June 26, 1955. Dr. Dille was a general practitioner.

★**William E. Dodsworth** (COL '15), 62, San Benito, Texas, died July 8, 1955. Dr. Dodsworth, who served for several years with the U. S. Bureau of Animal Industry, was admitted to the AVMA in 1929. He is survived by his widow.

Alfred J. DuFrene (MCK '10), Harlowton, Mont., died (date not known). Dr. DuFrene was a general practitioner.

Thomas B. Galbraith (MCK '02), Elmhurst, Ill., died recently. Dr. Galbraith was a general practitioner.

Gordius P. Gant (UVC '03), Trenton, Mo., died recently. Dr. Gant was a general practitioner.

Donald W. Hart (ISC '50), 35, Winthrop, Iowa, died in July, 1955. Dr. Hart was a general practitioner.

Charles C. Hofflin (BRN '22), Dawson Creek, B. C., died recently. Dr. Hofflin was a general practitioner.

Walter L. Howden (ONT '08), Kitchener, Ont., died Feb. 22, 1955. Dr. Howden is survived by his widow and two daughters.

R. A. Hughes (ONT '13), Orangeville, Ont., died Aug. 5, 1955.

Albert W. Judd (ONT '12), Lansing, Mich., died recently. Dr. Judd had served with the state bureau of animal industry.

Frederick M. Kettner (NYA '03), Staten Island, N. Y., died during August, 1953. Dr. Kettner was a general practitioner. He is survived by his widow.

★**William E. Kreider** (ONT '94), 83, Wadsworth, Ohio, died July 22, 1955. Dr. Kreider had been a member of the AVMA for 50 years. He is survived by his widow, four daughters, four grandchildren, and three great-grandchildren.

R. E. Kyner (ISC '11), Humeston, Iowa, died May 6, 1955.

F. G. Livingston (USC '14), 72, Newcastle, Neb., died in April, 1955. Dr. Livingston had practiced in Newcastle for 40 years. He is survived by his widow, four sons, a daughter, and seven grandchildren.

James E. Masterson (AVC '97), East Norwich, N. Y., died recently.

George A. McLevey (ONT '01), Rodney Elgin, Ont., died (date not known). Dr. McLevey, a general practitioner, had retired.

A. W. Miller (KCV '01), 79, Washington, D. C., died Sept. 1, 1955. An obituary appears on page 382 of this JOURNAL.

Dan M. Morrison (ONT '14), Edmonton, Alta., died in 1953. Dr. Morrison had been employed by the Edmonton City Health Department.

Temple H. Naylor (WSC '26), Wellsville, Kan., died recently. Dr. Naylor had specialized in swine practice.

Harold F. Newton (COL '41), 39, Adams City, Colo., died recently. Dr. Newton had been a member of the AVMA.

Charles A. Van Ausdall (CVC '12), Edinburg, Ill., died (date not known). Dr. Van Ausdall, a general practitioner, had also done state and federal work. He had been a member of the AVMA. His widow survives.

George B. Winch (CVC '14), 66, George, Iowa, died June 28, 1955. Dr. Winch had practiced in George for 42 years and had been active in community activities. He is survived by his widow, two daughters, and three grandchildren.

Edward L. Young (KCV '12), Grandview, Mo., died May 2, 1955. Dr. Young had been a member of the AVMA.

★Indicates members of the AVMA.

American Veterinary Medical Association
Official Roster, 1955-1956

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*This committee is charged with the selection of recipients of the Twelfth International Veterinary Congress Prize, the Borden Award, and the AVMA Award.

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Floyd Cross, *Chairman,* School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo.
Wayne O. Kester, Veterinary Service, Office of the Surgeon General, Department of Air Force, Washington 25, D.C.
Joseph M. Arburua, 3020-26th Ave., San Francisco, Calif.
J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill.
H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill.

Ethics

F. R. Booth, *Chairman,* 3920 E. Jackson St., Elkhart, Ind. (1958).
Fred Schell, P.O. Box 135, Franklin, Tenn. (1957).
E. L. Taylor, Warrendale Ave., Georgetown, Ky. (1956).

Council on Education

Niel W. Pieper,* *Chairman, Representing General Practice,* Randolph Rd., Middletown, Conn. (1958).
R. E. Rebrassier,* *Secretary, Representing Basic Sciences,* Veterinary Laboratory, Ohio State University, Columbus 10, Ohio (1960).
I. B. Boughton, *Representing Research and Education,* School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1958).
Garth A. Edge, *Representing Public Health,* Provincial Department of Public Health, Toronto, Ont. (1957).
James E. Green,* *Representing Clinical Sciences,* School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. (1956).
Col. William E. Jennings, *Representing Military Service,* Medical Field Service School, Fort Sam Houston, Texas (1956).
Fred B. Ogilvie, *Representing Small Animal Practice,* 1110-12 Minnesota Ave., Kansas City 2, Kan. (1960).
M. S. Shahan, *Representing Government Service,* Plum Island Animal Disease Laboratory U.S.D.A., Greenport, L.I., N.Y. (1961).
E. E. Slatter, *Representing Large Animal Practice,* 205 Prairie St., Danville, Ill. (1959).

*These three members compose the Executive Committee of the Council and are elected by the Executive Board; the remaining members are appointed by the president.

Food Hygiene

C. H. Pals, *Chairman,* Meat Inspection Branch, Agricultural Research Service, U.S.D.A., Washington, 25, D.C. (1958).
J. W. Cunkelman, Veterinary Division, Research Department, Swift and Company, Chicago, Ill. (1958).
Raymond J. Helvig, Milk and Food Section, Sanitary Engineering Division, U.S. Public Health Service, Washington 25, D.C. (1957).
Norman A. Fish, Ontario Veterinary College, Guelph, Ont. (1956).
John L. Owens, Station Veterinarian, 6006 Service Unit, Fort Lewis, Wash. (1956).
E. J. Rigby, City Health Department, Winnipeg, Man. (1957).

History

H. P. Hoskins, *Chairman,* 2766 Garrison Ave., Evanston, Ill. (1958).
Joseph M. Arburua, 3020-26th Ave., San Francisco, Calif. (1958).
R. R. Dykstra, School of Veterinary Medicine, Kansas State College, Manhattan, Kan. (1957).
C. D. Grinnells, Department of Animal Industry, North Carolina State College, Raleigh, N. Car. (1956).
Robert S. MacKellar, Sr., 329 W. 12th St., New York 14, N.Y. (1957).
Charles Murray, Rt. 3, Box 9, Santa Fe, N. M. (1956).

Joint Committee on Foods

Louis A. Corwin, *Chairman,* 136-21 Hillside Ave., Richmond Hill 18, N.Y. (AAHA) (1958).
J. G. Hardenbergh, *Secretary,* 600 S. Michigan Ave., Chicago 5, Ill. (AVMA) (1956).
C. W. Bower, 1128 Kansas Ave., Topeka, Kan. (AAHA) (1957).
J. B. Engle, P.O. Box 432, Summit, N. J. (AVMA) (1959).
Lloyd C. Moss, Veterinary Hospital, Colorado A. & M. College, Fort Collins, Colo. (1960).

Legislation

C. F. Clark, *Chairman,* School of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1958).

J. G. Hardenbergh, *Secretary, ex officio,* 600 S. Michigan Ave., Chicago 5, Ill.
E. A. Davis, P.O. Box 1534, Columbus, Ga. (1956).
Howard W. Johnson, Animal Disease Station, Beltsville, Md. (1957).
N. J. Miller, P.O. Box 335, Eaton, Colo. (1958).
O. H. Person, 359 N. Linden St., Wahoo, Neb. (1957).
Elmer W. Young, Veterinary Division, Office of the Surgeon General, Department of the Army, Washington 25, D.C. (1956).

Nutrition

William D. Pounden, *Chairman,* 625 Sunrise View Dr., Wooster, Ohio (1957).
Robert W. Dougherty, New York State Veterinary College, Ithaca, N. Y. (1957).
Raymond T. Hander, Bridwell Hereford Ranch, Windthorst, Texas (1956).
R. E. Lubbehusen, Purina Mills, 835 S. 8th St., St. Louis 2, Mo. (1956).
R. H. Udall, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo. (1957).
C. K. Whitehair, Department of Animal Husbandry, Oklahoma A. & M. College, Stillwater, Okla. (1956).

Parasitology

Leonard E. Swanson, *Chairman,* 1325 N.W. 14th Ave., Gainesville, Fla. (1958).
W. S. Bailey, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. (1956).
A. H. Groth, College of Veterinary Medicine, University of Missouri, Columbia, Mo. (1956).
Wendell Krull, School of Veterinary Medicine, Oklahoma A. & M. College, Stillwater, Okla. (1957).
Lee Seghetti, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo. (1957).
John H. Whitlock, New York State Veterinary College, Cornell University, Ithaca, N.Y. (1958).

Poultry Diseases

C. D. Lee, *Chairman,* B-12 Curtiss Hall, Iowa State College, Ames, Iowa (1958).
C. W. Barber, Department of Poultry Science, North Carolina State College, Raleigh, N. Car. (1956).
J. P. Delaplane, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1956).
C. L. Nelson, Jewell, Iowa (1958).
B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, Minn. (1957).
Frank C. Tucker, Claypool, Ind. (1957).

Program*

(Ex Officio)

This committee is composed of the chairmen and secretaries of the six sections with the executive secretary or assistant executive secretary acting as the chairman (*see* Section Officers, page 388).

*Pursuant to article XII, section I, part 4, of the Administrative By-Laws, as amended at the Seventy-Eighth Annual Meeting.

Public Relations

J. R. Pickard, *Chairman,* St. Joseph, Ill. (1958).
Dan J. Anderson, Rt. 1, Box 123, Smithfield, Texas (1958).
C. E. DeCamp, Pitman-Moore Co., 43 W. 61st St., New York 23, N.Y. (1956).
Kenneth G. McKay, 2086 Veterinary Science Bldg., University of California, Davis, Calif. (1956).
H. C. Smith, 2415 W. Solway, Sioux City, Iowa (1956).
Myron A. Thom, 959 S. Raymond Ave., Pasadena 2, Calif. (1957).

Registry of Veterinary Pathology Armed Forces Institute of Pathology

H. R. Seibold, *Chairman,* School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. (1956).
C. L. Davis, Bldg. 45, Denver Federal Center, Denver 1, Colo. (1958).
Hugh G. Grady, Scientific Director, American Registry of Pathology, Armed Forces Institute of Pathology, Washington 25, D.C. (consulting member).
T. C. Jones, Armed Forces Institute of Pathology, 7th St. and Independence Ave., S.W., Washington, D.C. (1955).
Charles A. Mitchell, Animal Disease Research Institute, Hull, Que. (1957).
Marvin Twiehaus, 700 Harris St., Manhattan, Kan. (1957).

Research Council

(Appointments are for three-year terms)

Anatomy and Histology.—Robert Getty, *Secretary,* Department of Anatomy, Iowa State College, Ames, Iowa (1958).
Bacteriology (Immunology and Biological Therapy).—L. C. Ferguson, *Chairman,* Department of Veterinary Science, Ohio Agricultural Experiment Station, Wooster, Ohio (1956).
Biochemistry and Animal Nutrition.—M. J. Swenson, Department of Physiology, School of Veterinary Medicine, Kansas State College, Manhattan, Kan. (1956).
Large Animal Medicine.—E. F. Ebert, School of Veterinary Medicine, University of Missouri, Columbia, Mo. (1956).
Large Animal Surgery.—A. Gordon Danks, New York State Veterinary College, Cornell University, Ithaca, N. Y. (1957).
Parasitology.—R. D. Turk, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1957).
Pathology.—T. Lloyd Jones, Ontario Veterinary College, Guelph, Ont. (1956).
Physiology and Pharmacology.—D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa. (1956).
Poultry Pathology.—C. A. Brandly, Department of Veterinary Science, University of Wisconsin, Madison, Wis. (1957).
Radiology.—Myron A. Thom, 959 S. Raymond Ave., Pasadena 2, Calif. (1957).
Small Animal Medicine.—Richard L. Ott, 1405 Gary St., Pullman, Wash. (1958).

Small Animal Surgery.—James Archibald, Ontario Veterinary College, Guelph, Ont. (1958).
Veterinary Hygiene.—A. G. Karlson, Mayo Foundation, Rochester, Minn. (1958).
Virology.—C. H. Cunningham, School of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1958).
Member-at-Large.—Burton J. Gray, P.O. Box 797, Fort Dodge, Iowa (1957).

Resolutions

(Appointments are for one-year terms)

W. W. Armistead, *Chairman,* School of Veterinary Medicine, Texas A. & M. College, College Station, Texas.
J. G. Hardenbergh, *Secretary, ex officio,* 600 S. Michigan Ave., Chicago 5, Ill.
R. McG. Archibald, 400 Prince St., Truro, N. S.
Ralph L. West, 310 Globe Bldg., St. Paul, Minn.
L. T. Hopkins, 23 Federal Bldg., Kansas City 2, Kan.

Subcommittee on Veterinary Items, National Formulary Committee

(Ten-year appointments terminating in 1959)

B. V. Alfredson, *Chairman,* School of Veterinary Medicine, Michigan State University, East Lansing, Mich.
D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa.
L. Meyer Jones, Division of Veterinary Medicine, Iowa State College, Ames, Iowa.
F. J. Kingma, Abbott Laboratories, North Chicago, Ill.
John E. Martin, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

Therapeutic Agents

L. Meyer Jones, *Chairman,* Division of Veterinary Medicine, Iowa State College, Ames, Iowa (1958).
Anthony R. Bott, 102 Osage Dr., Collinsville, Ill. (1957).
John H. Collins, Food and Drug Administration, Department of Health, Education, and Welfare, Washington 25, D.C. (1956).
D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa. (1958).
L. A. Gendreau, 143 Wellington St. S., Sherbrooke, Que. (1957).
Roger P. Link, Department of Veterinary Physiology and Pharmacology, College of Veterinary Medicine, University of Illinois, Urbana, Ill. (1956).

Special Committees

Academic Standards Board for Graduates of Foreign Veterinary Colleges

W. T. S. Thorp, *Chairman,* School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn.
John D. Gadd, Falls Road, Cockeysville, Md.
Ernest O. Froelich, 11 Edgewood Ave., Albany, N.Y.
William E. Jennings, Medical Field Service School, Fort Sam Houston, Texas.
T. Lloyd Jones, Ontario Veterinary College, Guelph, Ont.
Frantisek Kral, 838 Upland Ave., Upland-Chester, Pa.

Animal Reproduction and Artificial Insemination

David E. Bartlett, *Chairman,* American Breeders Service, 325 N. Wells St., Chicago 10, Ill.
William H. Dreher, Shawano, Wis.
Myron G. Fincher, 118 Delaware Ave., Ithaca, N.Y.
A. H. Frank, 4100 Roanoke Rd., Hyattsville, Md.
Harold J. Hill, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo.
E. A. Woelffer, 115 Woodland Lane, Oconomowoc, Wis.

Brucellosis

J. L. McAuliff, *Chairman,* 11 Jewett Ave., Cortland, N.Y.
Hugh S. Cameron, School of Veterinary Medicine, University of California, Davis, Calif.
Francis H. Fox, New York State Veterinary College, Ithaca, N.Y.
A. K. Kuttler, Animal Disease Eradication Branch, Agricultural Research Service, U.S.D.A., Washington 25, D.C.
A. M. Orum, P.O. Box 355, Carthage, Ill.
Leonard A. Rosner, Schumate Chapel Rd., Jefferson City, Mo.
V. D. Stauffer, 5500 Wadsworth Blvd., Arvada, Colo.

Diseases of Wild and Furbearing Animals

John R. Gorham, *Chairman,* Department of Animal Pathology, College of Veterinary Medicine, State College of Washington, Pullman, Wash.
T. T. Chaddock, Box 51, Bridgeport, Mich.
G. R. Hartsough, New Holstein, Wis.
R. J. Kirk, Fur and Game Experiment Station, University of Manitoba, Fort Garry, Man.
Patricia O'Connor, 97 Board St., Stapleton, Staten Island, N.Y.

Emergency Advisory Committee

W. R. Krill, *Chairman,* College of Veterinary Medicine, Ohio State University, Columbus, Ohio.
H. E. Kingman, Jr., *Secretary,* 600 S. Michigan Ave., Chicago 5, Ill.
J. B. Engle, P.O. Box 432, Summit, N. J.
W. R. Hinshaw, 140 Kline Blvd., Frederick, Md.
Frank A. Todd, Agricultural Research Service, U.S.D.A., Washington 25, D.C.
C. D. Van Houweling, Livestock Regulatory Programs, Agricultural Research Service, U.S.D.A., Washington 25, D.C.
Elmer W. Young, Veterinary Division, Office of the Surgeon General, Department of the Army, Main Navy Bldg., Washington 25, D.C.
Ex Officio: Board of Governors.

Humane Act Award

W. A. Young, *Chairman*, 8344 Beverly Blvd., Hollywood 48, Calif.
George W. Mather, School of Veterinary Medicine, University Farm, St. Paul 1, Minn.
Gerry B. Schnelle, 180 Longwood Ave., Boston, Mass.
Mr. J. J. Shaffer, *Advisory Member*, 157 W. Grand Ave., Chicago 10, Ill.
A. R. Theobald, 4545 Reading Rd., Cincinnati 29, Ohio.

Insurance*

O. Norling-Christensen, *Chairman*, 730 Hibbard Rd., Wilmette, Ill.
Carl A. Brandly, Department of Veterinary Science, University of Wisconsin, Madison, Wis.
Homer D. Carter, 410 N. Main St., Fairmount, Ind.

*Remainder of committee to be appointed in accordance with recommendations of the 1955 session of the House of Representatives.

Laboratory Animals

N. R. Brewer, *Chairman*, Physiology Bldg., University of Chicago, Chicago 37, Ill.
Robert C. Bay, College of Medicine, University of Utah, Salt Lake City, Utah.
Jules S. Cass, Kettering Laboratory, College of Medicine, Eden Ave., Cincinnati 19, Ohio.
W. H. Dieterich, Hawaiian Medical Laboratory, APO 957, San Francisco, Calif.
R. J. Flynn, P.O. Box 299, Lemont, Ill.
R. Dale Henthorne, Army Medical Service Graduate School, Veterinary Division, Walter Reed Army Medical Center, Washington 12, D.C.

Motion Pictures and Television

C. D. Van Houweling, *Chairman*, Livestock Regulatory Programs, Agricultural Research Service, U.S.D.A., Washington 25, D.C.
Robert Getty, Division of Veterinary Medicine, Iowa State College, Ames, Iowa.
L. J. Gorman, 1540 Ostrander St., La Grange Park, Ill.
J. A. Henderson, Ontario Veterinary College, Guelph, Ont.
C. L. Nelson, Jewell, Iowa.
Wayne H. Riser, 5335 Touhy Ave., Skokie, Ill.
Wayne D. Shipley, 438 S. Quincy St., Hinsdale, Ill.
Frank A. Todd, Agricultural Research Service, U.S.D.A., Washington 25, D.C.

Nomenclature of Diseases

Carl Olson, Jr., *Chairman*, Deparment of Animal Pathology and Hygiene, University of Nebraska, Lincoln, Neb.
C. Lawrence Blakely, Angell Memorial Animal Hospital, 180 Longwood Ave., Boston, Mass.
D. H. Clifford, School of Veterinary Medicine, University of Minnesota, University Farm, St. Paul 1, Minn.
D. E. Jasper, School of Veterinary Medicine, University of California, Davis, Calif.
I. A. Merchant, Division of Veterinary Medicine, Iowa State Colege, Ames, Iowa.
Peter Olafson, New York State Veterinary College, Ithaca, N. Y.

Public Health and Zoonoses

E. S. Tierkel, *Chairman*, Communicable Disease Center, U. S. Public Health Service, 50 7th St. N.E., Atlanta 5, Ga.
William S. Gochenour, Jr., Research and Graduate School, Veterinary Division, Army Medical Center, Washington 12, D. C.
R. W. Menges, Department of Health, Education, and Welfare, Communicable Disease Center, P.O. Box 185, Chamblee, Ga.
I. A. Merchant, Division of Veterinary Medicine, Iowa State College, Ames, Iowa.
Karl R. Reinhard, Arctic Health Research Center, P.O. Box 960, Anchorage, Alaska.
J. H Steele, Communicable Disease Center, U. S. Public Health Service, 50 7th St. N.E., Atlanta 5, Ga.
Kenneth F. Wells, 506 Piccadilly Ave., Ottawa, Ont.

Scientific Exhibits

Asa Winter, *Chairman*, 1612 N. Jefferson St., Arlington 5, Va.
E. A. Benbrook, Department of Veterinary Pathology, Iowa State College, Ames, Iowa.
N. R. Brewer, Physiology Bldg., University of Chicago, Chicago 37, Ill.
Charles L. Davis, Bldg. 45, Denver Federal Center, Denver 1, Colo.
T. C. Jones, Armed Forces Institute of Pathology, 7th St. and Independence Ave., S.W., Washington, D. C.

Standards for Veterinary Hospitals

Kenneth W. Smith, *Chairman*, 1002 34th St., Sioux City, Iowa.
Charles E. Bild, P.O. Box 127, North West Station, Miami, Fla.
A. F. Hayes, P. O. Box 1703, Billings, Mont.
H. L. Marsh, 521 Billings St., Princeton, Ill.
L. C. Moss, Veterinary Hospital, Colorado A. & M. College, Fort Collins, Colo.

Veterinary Service

I. D. Wilson, *Chairman*, Virginia Polytechnic Institute, Blacksburg, Va.
H. E. Kingman, Jr., *Secretary*, 600 S. Michigan Ave., Chicago 5, Ill.
F. T. Candlin, 280 Madison St., Denver 6, Colo.
Lewis A. Dykstra, 925 N. Lake St., Aurora, Ill.
E. J. Frick, School of Veterinary Medicine, Kansas State College, Manhattan, Kan.
Warren J. Kilpatrick, Mediapolis, Iowa.
Charles C. Rife, 420 Edgewood Ave., N.E., Atlanta 3, Ga.

Veterinary Supply Problems

F. B. Young, *Chairman*, Box 6, Waukee, Iowa.
R. O. Anderson, 223 N. Wisconsin St., Elkhorn, Wis.
P. E. Madsen, 336 N. Jefferson St., Sheridan, Wyo.
Hadleigh Marsh, Blackstone Apts., Helena, Mont.
A. G. Misener, 6448 N. Clark St., Chicago, Ill.
Kenneth Whittington, 769 Vance Ave., Memphis, Tenn.

Representatives

(Terms are for one year except as shown)

American Association for the Advancement of Science.—W. H. Feldman, 926 8th Ave. S.W., Rochester, Minn. (1955).

American Feed Manufacturers Association Council on Nutrition. — William D. Pounden, 625 Sunrise View Dr., Wooster, Ohio.

American Medical Association Council on National defense.—H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill.

Livestock Conservation Inc.—H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill.

National Brucellosis Committee.—A. M. Orum, Box 355, Carthage, Ill.

National Health Council.—J. A. McCallam, Rm. 109, 1507 M. St., N.W., Washington 5, D.C.

National Research Council (Division of Biology and Agriculture).—E. F. Waller, Department of Animal and Poultry Industry, University of Delaware, Newark, Del. (1957).

National Research Council, Division of Medical Sciences.—W. A. Hagan, New York State Veterinary College, Ithaca, N. Y. (1956).

National Society for Medical Research.—W. T. S. Thorp, School of Veterinary Medicine, University of Minnesota, University Farm, St. Paul 1, Minn. (1956).

Ralston Purina Research Fellowship Committee.—L. C. Ferguson, Department of Veterinary Science, Ohio Agricultural Experiment Station, Wooster, Ohio (1956).

United States Pharmacopoeial Convention.—D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa. (1960).

Resident State Secretaries

Alabama.—McKenzie Heath, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn.

Arizona.—James E. Sharkey, 6021 S. Central Ave., Phoenix.

Arkansas.—W. L. Thomas, 906 Broadway, Little Rock.

California.—E. E. Jones, 714 S. Santa Anita St., San Gabriel.

Colorado.—Robert K. Anderson, 860 Olive St., Denver.

Connecticut.—Niel W. Pieper, Randolph Rd., Middletown.

Delaware.—E. F. Waller, Department of Animal and Poultry Industry, University of Delaware, Newark.

District of Columbia.—William C. Patterson, Jr., Animal Disease Station, Beltsville, Md.

Florida.—Stanley C. Wasman, 1929 Purdy Ave., Miami Beach.

Georgia.—Charles C. Rife, 420 Edgewood Ave. N.E., Atlanta 3.

Idaho.—A. P. Schneider, 108 Capitol Bldg., Boise.

Illinois.—C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines.

Indiana.—John L. Kixmiller, 4144 N. Capitol Ave., Indianapolis 8.

Iowa.—F. B. Young, P.O. Box 6, Waukee.

Kansas.—K. M. Curts, 70 Central Ave., Kansas City.

Kentucky.—T. J. Stearns, 216 Livestock Exchange Bldg., Louisville 6.

Louisiana.—R. B. Lank, Dalrymple Bldg., Louisiana State University, Baton Rouge.

Maine.—Harold L. Chute, Animal Pathology Bldg., University of Maine, Orono.

Maryland.—John D. Gadd, Falls Rd., Cockeysville.

Massachusetts.—L. A. Paquin, P.O. Box 225, Webster.

Michigan.—Paul V. Howard, 4011 Hunsberger Dr. N.E., Grand Rapids 5.

Minnesota.—Alvin F. Weber, School of Veterinary Medicine, University of Minnesota, University Farm, St. Paul 1.

Mississippi.—Charles H. Horne, P.O. Box 377, Newton.

Missouri.—A. H. Groth, College of Veterinary Medicine, University of Missouri, Columbia.

Montana.—A. M. Jasmin, Livestock Sanitary Board, Helena.

Nebraska.—Paul L. Matthews, 4901 S. 33rd St., Omaha.

Nevada.—Edward Records, University of Nevada, Reno.

New Hampshire.—Clarence F. Bent, Stateline-Lowell Rd., Nashua.

New Jersey.—J. R. Porteus, P.O. Box 938, Trenton 5.

New Mexico.—S. W. Wiest, P.O. Box 75, Santa Fe.

New York.—Charles E. Fanslau, 1450 Broadway, New York 18.

North Carolina.—C. W. Young, 203 Depot St., Mocksville.

North Dakota.—S. S. Bjornson, Union Stockyards, West Fargo.

Ohio.—Harold F. Groves, 111 E. Kelso Rd., Columbus 2.

Oklahoma.—C. H. Fauks, 3421 N.W., 20th St., Oklahoma City.

Oregon.—Edward L. Holden, P.O. Box 445, Oswego.

Pennsylvania.—Roy D. Hoffman, 325 S. Richard St., Bedford.

Rhode Island.—J. S. Barber, 560 Pleasant St., Pawtucket.

South Carolina.—Frank M. Lee, State Board of Health, Wade Hampton State Office Bldg., Columbia.

South Dakota.—J. L. Noordsy, Marion.
Tennessee.—Hugh Lamb, P.O. Box 550, Athens.
Texas.—L. G. Cloud, 2833 W. 7th St., Fort Worth.
Utah.—Hugh Hurst, 1754 E. 27th St. S., Salt Lake City.
Vermont.—A. E. Janawicz, R.F.D. 2, Montpelier.
Virginia.—Wilson B. Bell, 210 Clay St., Blacksburg.
Washington.—J. L. Ellis, 2022 E. 4th Ave., Olympia.
West Virginia.—Victor H. Miller, Box 2881, Charleston 21.
Wisconsin.—H. J. O'Connell, 6 West, State Capitol, Madison 2.
Wyoming.—J. F. Ryff, P.O. Box 557, Laramie.

Resident Provincial Secretaries

Alberta.—J. Gordon Anderson, 1016 9th Ave. W., Calgary.
British Columbia.—Gordon L. Davis, P.O. Box 32, Ladner.
Manitoba.—J. M. Isa, Veterinary Laboratory, University of Manitoba, Winnipeg.
New Brunswick.—Julius F. Frank, Division of Animal Pathology, P.O. Box 310, Sackville.
Nova Scotia.—R. McG. Archibald, 400 Prince St., Truro.
Ontario.—W. J. Rumney, Health Center, 74 Hughson St. S., Hamilton.
Quebec.—Roland Filion, Laboratoire de Recherches Veterinaires, Saint-Hyacinthe.
Saskatchewan.—J. S. Fulton, University of Saskatchewan, Saskatoon.

Resident Territorial Secretaries

Canal Zone.—Robert G. Matheney, P.O. Box 807, Ancon.
Hawaii.—Wilson M. Pang, 89 Ala Kimo, Honolulu 17.
Puerto Rico.—A. Lopez-Pacheco, P.O. Box 155, Hato Rey.

Correspondents

Alaska.—Earl F. Graves, Box 517, Spenard.
Argentina.—Carlos T. Rosenbusch, San Jose 1481, Buenos Aires.
Australia.—Colin T. Petherbridge, 47 Sunderland St., Mayfield.
Azores.—Jose L. Armas, Rua da Rainha D Amelia 121-2, Angra do Heroismo Terceira.
Bahamas.—Albert Soltys, P.O. Box 1234, Nassau.
Bermuda.—John W. Sutherland, Fairylands, Pembroke W.
Brazil.—A. V. Machado, Rua Paracatu 1.178, Belo Horizonte.
British West Africa.—Desmond H. Hill, University College, Ibadan, Nigeria.
British West Indies.—Stephen P. Bennett, "Glamorgan," Gordon St., Curepe, Trinidad.
Ceylon.—Don W. Amarasinghe, Municipal Veterinary Surgeon, Colombo.
Chile.—Arturo Hernandez, Dardignac 95, Santiago.
Colombia.—Juan A. Villamil, Cra. 14 68-91, Bogota.
Cuba.—Angel M. Morales, Calle 30 No. 310, Vedado, Havana.
Denmark.—N. Ole Rasbech, Royal Veterinary and Agricultural College, Bulowsvej 13, Cophenhagen V.
Equador.—Julio Bolona R., Casilla 3814, Guayaquil.
Egypt.—J. E. Aghion, 20 Sharia Senan, Pasha, Zeitoun.
Erie.—P. A. Rogan, Knockeevin Church Rd., Greystones, County Wicklow.
El Salvador.—Miguel A. Sandoval, Avenida Araujo 23, San Salvador.
England.—Guy Anderson, Aylesbury, Buckinghamshire.
Formosa (Free China).—Sou-Shen Young, Department of Veterinary Medicine, College of Agriculture, National Taiwan University, Taipeh, Taiwan.
Guatemala.—Francisco R. Rodas C., 7a. Avenida 5-61 Zona 4, Guatemala City.
Honduras.—Fernando J. Gonzalez, Tela Railroad Co., Progresso.
India.—Bimolendu Choudhury, 14-B Park Side Rd., Calcutta 26.
Israel.—Joachim Weis, Moledeth Ein Charod.
Italy.—L. Taglia, via Lucania 9, Rome.
Japan.—Kogi Saito, 69 4-Chrome Fukazawamachi, Setagayaku, Tokyo.
Mexico.—Alfonso Alexander H., Insurgentes No. 458, Mexico, D.F.
New Zealand.—Daniel J. Smith, P.O. Box 85, Putaruru.
Panama Republic.—A. A. Arosemena, P. O. Box 192, Panama City.
Peru.—Juan A. Pinto C., Jiron Huaraz 1313 (Brena), Lima.
Philippines, Republic of.—Jose B. Aranez, College of Veterinary Medicine, University of the Philippines, Diliman, Quezon City.
Prince Eward Island.—J. R. Cunningham, Summerside.
Spain.—Juan Talavera, Calle Iturbe 14, Madrid.
Sweden.—Fritz Magnus Nilsson-Sevelius, Tradgardsgatan 3, Halsingborg.
Thailand.—Choomphorn Gomutputra, 202 Rajavithi Rd., Vajira Pyabal District, Bankok.
Turkey.—Namik Buharalilar, Ahmetfaki Mahallesi No. 10, Konya.
Union of South Africa.—John G. Townsend, 172 Jan Smutz Ave., Rosebank, Johannesburg, Transvaal.
Uruguay.—G. P. Lockhart, Av. Italia 2538, Montevideo.
Venezuela.—Claudio E. Muskus, P. O. Box 993, Alesandro Divo, Caracas.

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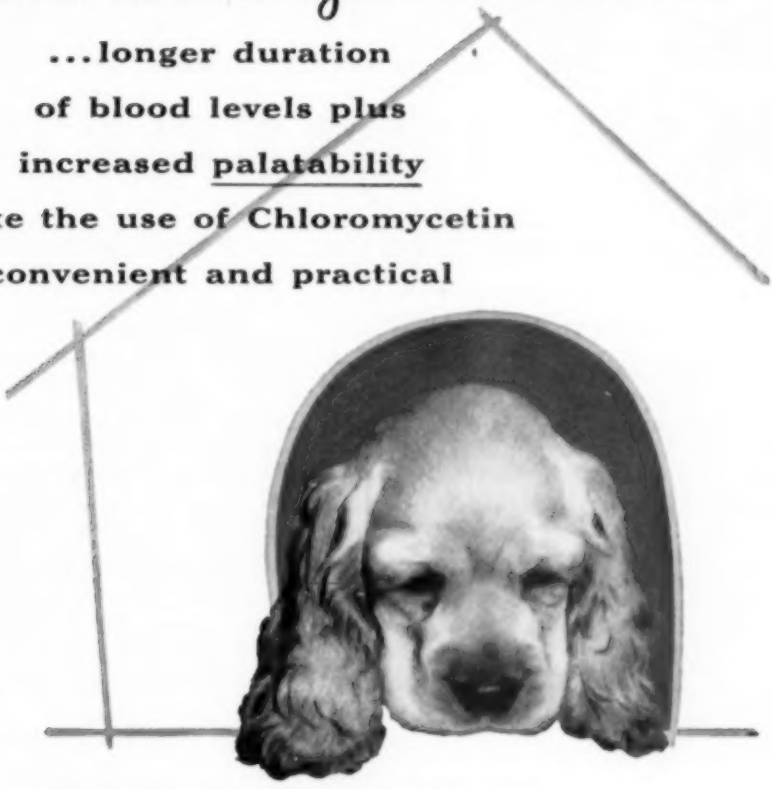

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Suspension Chloromycetin Palmitate (suspension chloramphenicol Parke-Davis) is supplied in 60 cc. bottles, each 4 cc. (teaspoonful) representing 125 mg. of Chloromycetin.

PROFESSIONAL LITERATURE AVAILABLE ON REQUEST

Department of Veterinary Medicine

PARKE, DAVIS & COMPANY

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COMING MEETINGS

Notices of Coming Meetings must be received by 4th of month preceding date of issue

New England Veterinary Medical Association. Annual meeting. Poland Spring House, Poland Spring, Maine, Oct. 2-5, 1955. Russell N. Abbott, Rockland, Maine, publicity chairman.

Missouri, University of. Annual short course. School of Veterinary Medicine, University of Missouri, Oct. 3-4, 1955. Cecil Elder, chairman, Veterinary Short Course Committee.

International Association of Milk and Food Sanitarians. Annual meeting. Hotel Bon-Air, Augusta, Ga., Oct. 4-6, 1955. Howard Wilkowske, Gainesville, Fla., secretary.

Purdue University. Annual short course for veterinarians. Purdue University, Lafayette, Ind., Oct. 5-7, 1955. L. M. Hutchings, head, Department of Veterinary Medicine.

West Virginia Veterinary Medical Association. Annual meeting. West Virginian Hotel, Bluefield, W. Va., Oct. 9-10, 1955. D. A. Munro, 202 Oglebay Hall, West Virginia University, Morgantown, W. Va., secretary.

District of Columbia Veterinary Medical Association. All-day meeting. Armed Forces Institute of Pathology, Walter Reed Medical Center, Washington, D. C., Oct. 11, 1955. Charles G. Durbin, 5705 Berwyn Rd., Berwyn Heights, College Park, Md., secretary.

South Dakota Veterinary Medical Association. Annual meeting. Cataract Hotel, Sioux Falls, S. Dak., Oct. 12-13, 1955. J. L. Noordsy, Marion, S. Dak., secretary.

Eastern Iowa Veterinary Medical Association, Inc. Annual meeting. Hotel Montrose, Cedar Rapids, Iowa, Oct. 13-14, 1955. Wayne H. Thompson, Earlville, Iowa, secretary.

Illinois, University of. Annual short course for veterinarians. School of Veterinary Medicine, University of Illinois, Urbana, Ill., Oct. 13-14, 1955. L. E. Boley, chairman, Veterinary Conference Committee.

Southern Veterinary Medical Association and Florida Veterinary Medical Association. Joint annual meeting. George Washington Hotel, Jacksonville, Fla., Oct. 16-19, 1955. A. A. Husman, 320 Agricultural Bldg., Raleigh, N. Car., secretary, Southern Association.

Interstate Veterinary Medical Association. Annual meeting. Martin Hotel, Sioux City, Iowa, Nov. 1-2, 1955. K. W. Smith, 510 W. 19th St., Sioux City, Iowa, secretary.

Association of Military Surgeons of the United States. Annual convention. Hotel Statler, Washington, D. C., Nov. 7-9, 1955. Address Secretary, Suite 718, New Medical Bldg., 1726 Eye St., N. W., Washington 6, D. C.

Midwest Small Animal Hospital Association and regional A.A.H.A. Joint meeting. Hotel Burlington, Burlington, Iowa, Nov. 9-10, 1955. J. Porter Coble, 2828 S. MacArthur Blvd., Springfield, Ill., secretary.

U. S. Livestock Sanitary Association. Annual meeting. Jung Hotel, New Orleans, La., Nov. 16-18, 1955. R. A. Hendershott, 1 W. State St., Trenton 8, N. J., secretary.

Animal Care Panel. Annual meeting. Henry Hudson Hotel, 353 W. 57th St., New York, N. Y., Dec. 1-2, 1955. Robert J. Flynn, P.O. Box 299, Lemont, Ill., secretary.

Cornell University. Annual conference for veterinarians. New York State Veterinary College, Cornell University, Ithaca, N. Y., Jan. 4-6, 1956. W. A. Hagan, dean.

Oklahoma Veterinary Medical Association. Annual meeting. Hotel May, Tulsa, Okla., Jan. 8-10, 1956. C. H. Fauks, 3421 N.W. 20 St., Oklahoma City, Okla., secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin, Indianapolis, Ind., Jan. 11-13, 1956. L. M. Borst, 3315 Shelby Ave., Indianapolis, Ind., secretary.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Des Moines, Iowa, Jan. 17-19, 1956. F. B. Young, Waukee, Iowa, executive secretary.

Virginia Veterinary Medical Association. Annual meeting. Hotel John Marshall, Richmond, Va., Jan. 22-24, 1956. Wilson B. Bell, 210 Clay St., Blacksburg, Va., secretary.

California State Veterinary Medical Association. Midwinter conference. School of Veterinary Medicine, University of California, Davis, Calif., Jan. 23-25, 1956. Charles S. Travers, 3004 16th St., San Francisco, Calif., executive secretary.

Foreign Meetings

Tenth International Congress of Entomology. McGill University and University of Montreal, Montreal, Canada, Aug. 17-25, 1956. J. A. Downes, Division of Entomology, Science Service Bldg., Ottawa, Ont., Canada, secretary.

International Association of Hydatidology. Sixth Congress. Athens, Greece, Sept. 14-18, 1956. Prof. B. Kourias, 1 MacKenzie King St., Athens, Greece, general secretary.

(Continued on p. 32)

Blackhead mortality reduced from 50% to 10% or less with

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IN TURKEY BLACKHEAD . . .

Furoxone, the new antimicrobial nitrofuran recently introduced for treatment of avian salmonelloses (pullorum, typhoid, paratyphoids) is now established as exceptionally effective in prevention and treatment of Histomonas meleagridis infection (blackhead) of turkeys.[1,2]

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Furoxone proved superior to both an antibiotic and a sulfonamide in treating experimental Salmonella pullorum infections in chickens.[4] When therapy was started 1 day after infection, none of the Furoxone-treated chickens died, as compared with 90% deaths among untreated controls.

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Furoxone is also effective in fowl typhoid (S. gallinarum)[6] and paratyphoids (S. typhimurium et al.).

1. Harwood, P. D., and Stunz, D. I.: J. Parasitol. **40**: 24 (Oct.) 1954. 2. Pomeroy, B. S., and Pilkey, A. M.: Unpublished results. 3. Moore, E. N.; Chamberlin, V. D., and Carter, R. D.: Poultry Sci. **33**: 1072 (Sept.) 1954. 4. Smith, H. W.: Vet. Record **66**: 493 (Aug. 28) 1954. 5. Gordon, R. F., and Tucker, J.: Vet. Record **67**: 116 (Feb. 5) 1955. 6. Smith, H. W.: J. Comp. Path. **65**: 55 (Jan.) 1955.

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(COMING MEETINGS—*continued from p. 30*)

Regularly Scheduled Meetings

Atlanta (Ga.) Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. D. A. Buchanan, Grundy Center, Iowa, secretary.

Central Alabama Veterinary Association, the first Thursday of each month. G. J. Phelps, Jr., Montgomery, Ala., secretary.

Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Wilfred Pimentel, 3455 S. Elm Ave., Fresno, Calif., secretary.

Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel in Greensboro, N. Car. R. T. Copeland, 1800 Walker Ave., Greensboro, N. Car., secretary.

Central Indiana Veterinary Medical Association, the second Wednesday of each month. Charles J. York, P. O. Box 1656, Indianapolis 6, Ind., secretary.

Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., La-Grange, Ill., secretary.

Coastal Bend (Texas) Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Cuyahoga County (Cleveland, Ohio) Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

(*Continued on p. 34*)

East Bay (Calif.) Veterinary Medical Association, bimonthly, the fourth Wednesday. John T. Turver, 1201 E. 12th St., Oakland 6, Calif., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. R. P. Link, College of Veterinary Medicine, University of Illinois, Urbana, Ill., secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. John D. Baker, Goldsboro, N. Car., secretary.

Fayette County Veterinary Association, Iowa, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Greater St. Louis (Mo.) Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Luther E. Fredrickson, Room 25, Municipal Courts Bldg., St. Louis, Mo., secretary.

Jacksonville (Fla.) Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. L. D. Barrett, Rt. 8, Box 572, Jacksonville, Fla., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. Dr. W. E. Bewley, P.O. Box "H", Crestwood, Ky., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

Kern County (Calif.) Veterinary Medical Association, the first Thursday evening of each month. B. C. Watson, 825 14th St., Bakersfield, Calif., secretary.

Keystone (Pa.) Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St. W., Huntington, W. Va.

(*Continued on p. 36*)

(COMING MEETINGS—*continued from p. 34*)

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Michiana Veterinary Medical Association, the second Thursday of each month, at the Hotel LaSalle, South Bend, Ind. L. D. Ramsay, 719 E. Jefferson Ave., La Porte, Ind., secretary.

Michigan, Southeastern Veterinary Medical Association, the second Thursday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Road, Detroit 5, Mich., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. George McCollister, 2146 Broad St., San Luis Obispo, Calif., secretary.

Mid-State (Mich.) Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

Monterey Bay Area (Calif.) Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90, Corral de Tierra, Salinas, Calif., secretary.

New Castle County (Del.) Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. Arthur P. Coogan, 2102 New Road, Wilmington 5, Del., secretary.

New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northern Colorado Veterinary Medical Society, the second Monday of each month. William H. Beckenhauer, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck, N. J. Edward Baker, 568 Grand Ave., Englewood, N. J., secretary.

(*Continued on p. 38*)

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OUR NEW EXTRA LARGE

500-500 28cc MASTITIS TUBES

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IN 10 GROSS LOTS

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500,000 Units	Cryst. Penicillin Potassium G	53.28	4.44	37¢	10 gross
500 mg	Dihydrostreptomycin (as sulfate)	57.60	4.80	40¢	5 gross
750 mg	Sulfamerazine, Microcrystalline	61.92	5.16	43¢	3 gross
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7.5 mg	COBALT SULFATE	——	5.88	49¢	dozen

500-250 Each 11cc Liquid Base Tube Contains		Per Gross	Per Dozen	Per Tube	
500,000 Units	Cryst. Penicillin Potassium G	$38.80	$3.24	27¢	10 gross
250 mg	Dihydrostreptomycin (as sulfate)	43.20	3.60	30¢	5 gross
500 mg	Sulfamerazine, Microcrystalline	46.08	3.84	32¢	3 gross
500 mg	Sulfathiazole Microcrystalline	50.40	4.20	35¢	1 gross
7.5 mg	COBALT SULFATE		4.68	39¢	Dozen

300-100 Each 11cc Liquid Base Tube Contains					
300,000 Units	Cryst. Penicillin Potassium G	31.68	2.65	22¢	10 gross
100 mg	Dihydrostreptomycin (as sulfate)	36.00	3.00	25¢	5 gross
500 mg	Sulfamerazine, Microcrystalline	38.80	3.24	27¢	3 gross
500 mg	Sulfathiazole Microcrystalline	41.76	3.48	29¢	1 gross
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Northern San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month. Ernest Makino, Patterson, Calif., secretary.

Oklahoma County Veterinary Medical Association, the second Wednesday of every month except July and August. Carl L. Clark, 127 N. W. 23rd St., Oklahoma City, Okla., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month at 8:00 p.m. in Antlers Hotel, San Bernardino, Calif. Jay C. Wallis, 112 N. Girard St., Hemet, Calif., secretary.

Orange County (Calif.) Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula (Calif.) Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Piedmont (N. Car.) Veterinary Medical Association, the last Friday of each month at 7:00 p.m. in Mull's Motel in Hickory, N. Car. W. W. Dickson, Box 1071, Gastonia, N. Car., secretary.

Piedmont (S. Car.) Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

Pima County (Ariz.) Veterinary Medical Association, the third Wednesday of each month in Tucson. E. T. Anderson, 8420 Tanque Verde Rd., Tucson, Ariz., secretary.

Redwood Empire (Calif.) Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley (Calif.) Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

Saginaw Valley (Mich.) Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

San Diego County (Calif.) Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley (Calif.) Veterinary Medical Association, the second Friday of each month at Eaton's Restaurant in Studio City, Calif. R. A. Button, 5954 Van Nuys Blvd., Van Nuys, Calif., secretary.

Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R., Des Rosiers, 5508 2nd Ave. N. W., Seattle 7, Wash., secretary.

Southeastern (Mich.) Veterinary Medical Asso-

(*Continued on p. 40*)

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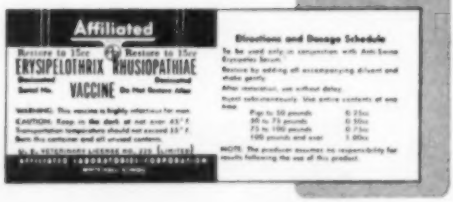

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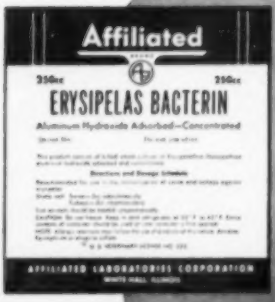

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An intermediate product ranking between serum alone, and simultaneous vaccination with serum-vaccine. Produces good immunity . . . Can be administered at the time of hog cholera vaccination when either serum and vaccine or serum and virus are used . . . can be used in states where use of vaccine isn't permitted.

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PRODUCED FOR AND SOLD TO GRADUATE VETERINARIANS ONLY

(COMING MEETINGS—*continued from p. 381*)

ciation, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

Southern California Veterinary Medical Association, the third Wednesday of each month. Howard C. Taylor, 2811 West Olive St., Burbank, Calif., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

South Puget Sound (Wash.) Veterinary Medical Association, the second Thursday of each month except July and August. Jo Walker, Agriculture Experiment Station, Puyallup, Wash., secretary.

Tenth District (Ind.) Veterinary Medical Association the third Thursday of each month. W. E. Sharp, Union City, Ind., secretary.

Tulare County (Calif.) Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Tulsa (Okla.) Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Merle S. Watts, 5302 E. 11th St., Tulsa, Okla., secretary.

Swift's Pard Meal has up to 50% more meat fat

FOR GREATER FLAVOR AND NUTRITION

No wonder dogs have shown a *2 to 1* preference for Pard Meal in actual feeding tests!

It tastes meatier . . . it *is* meatier! Swift puts *extra* amounts of meat fat in Pard Meal. And Pard Meal contains *every* food element a dog needs. In fact it's so rich in concentrated food value you can actually feed up to 25% *less* by volume!

Try it in your own kennels. Order it in 25 or 50-lb. bags.

Like Pard Meal, Swift's canned PARD is the result of years of research in Swift's laboratories and kennels. It completely eliminates the "guesswork" from the science of animal feeding. Pard is all a dog needs or wants, with GOOD BEEF TASTE dogs love!

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Veterinarian wanted with Florida license for mixed practice at small hospital in south Florida. Profit sharing arrangement or partnership to be worked out. Address "Box V 2," c/o JOURNAL of the AVMA.

Male veterinarian wanted as assistant in small animal practice in Ohio. Give age and personal data; salary plus commission offered. Address Dr. John W. Garling, 3209 Sylvania Ave., Toledo, Ohio.

Veterinarian wanted as associate in small animal hospital on Long Island. Very active practice with further room for expansion. Man must be experienced and capable of handling practice. Salary plus commission to start; advancement toward working partnership. New York State license required. Address "Box V 4," c/o JOURNAL of the AVMA.

Excellent opportunity for veterinarian with New York State license, assist in mixed practice on eastern Long Island. Good future, permanent basis. Address "Box V 5," c/o JOURNAL of the AVMA.

Veterinarian wanted to work for progressive Middlewest commercial company. Must have working knowledge of poultry and livestock under farm conditions. Attractive future for the right man. Address "Box V 6," c/o JOURNAL of the AVMA.

Research veterinarian wanted, experienced in biological production and professional services work; good future for right man in nationally known organization. Address "Box V 8," c/o JOURNAL of the AVMA.

Veterinarian wanted for poultry disease diagnostic laboratory with commercial organization in the East. Salary open. Address "Box V 9," c/o JOURNAL of the AVMA.

Veterinarian wanted for Livestock Disease Control State Laboratory in Boise, Idaho. Address Room 206, Statehouse, Boise, Idaho.

(Continued on p. 44)

From northern Japan comes this shipment of an ingredient for Purina Chows.

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PURINA'S search for the very highest quality ingredients goes all over the world. Often within a day's time our buyers will talk by long distance telephone or cable with Japan, Iceland, Norway and South Africa. They are on the job all the time, hunting for the best sources of highly essential ingredients.

This system of buying, coupled with our strict quality control program, gives us what we must have to build the kind of Chows that make feed dollars count.

Why does Purina go to the ends of the earth to buy these tiny but mighty ingredients? *Because we feel it is our responsibility to put into the Checkerboard bag the finest nutrition that modern science can provide.*

Yes, just like veterinary practice, animal nutrition is a science. Both are jobs for specialists. This fact is recognized by thousands of farm folks. They depend on their local veterinarian to keep disease and parasite losses to a minimum and on their Purina Dealer to supply sound feeding and management knowledge.

RALSTON PURINA COMPANY
St. Louis 2, Missouri

(CLASSIFIED ADS—*continued from p.* 42)

Young ambitious veterinarian looking for permanency in mixed practice wanted to do large animal work in well-established practice in Georgia. Very discriminating clientele, well versed, and demanding the best in service and diagnosis. State age, experience, marital status, and starting salary desired. Address "Box V 10," c/o JOURNAL of the AVMA.

Veterinarian needed to take over office and mixed practice of deceased veterinarian. Address "Box V 12," c/o JOURNAL of the AVMA.

Veterinarian with New York State license wanted as assistant in small animal practice on Long Island. State qualifications and salary expected. Address "Box T 2," c/o JOURNAL of the AVMA.

Veterinarian wanted for mixed practice in Connecticut. Salary to start; profit sharing and partnership in near future. Address "Box V 15," c/o JOURNAL of the AVMA.

(*Continued on p.* 46)

Cat alertness and appearance as affected by Good Nutrition

The known predilection of cats for fish finds interesting nutritive justification. Biochemists and biologists are generally agreed that fish—*whole* fish; not parts, scraps or offal—is the best single food a cat can eat.

Fish flesh proteins are believed to be equal or superior to those of beef for ease of digestion and maintenance of the body. Fish flesh, with its high proportion of essential unsaturated fatty acids, is highly digestible, hence easily absorbed and converted into energy. Fish minerals and vitamins, from fish bone structure, liver and glands, are similarly unique in their contribution to feline health, energy and beauty.

Puss 'n Boots Cat Food is made of whole fish with its beneficial balance of vital elements.

To the finely ground whole fish are added seven specially selected cereals, for all-round nutrition. Cats which have been inadequately fed show a remarkable improvement within three weeks on Puss 'n Boots. It is available to your clients at food stores and pet shops everywhere, in two sizes.

How the Natural Life Balance of WHOLE FISH is retained in PUSS 'n BOOTS

BONE STRUCTURE for calcium, phosphorus. Made digestible. *Retained in Puss 'n Boots.*

FILLETS OR FLESH, for proteins. Generally reserved for humans. *Retained in Puss 'n Boots.*

LIVER GLANDS. For vitamins A, B, D, and minerals. Essential for general well-being, healthy nerves. Often extracted for medical use. *Retained in Puss 'n Boots.*

If you have a camera, you will be interested in this exclusive new book by Walter Chandoha, world's foremost cat photographer. Beautifully designed and printed, "How to Photograph Your Cat" shows, for the first time Chandoha's professional techniques for posing cats, camera settings, etc. The makers of Puss 'n Boots Cat Food will be pleased to send you a copy without charge, upon receipt of a request on your professional letterhead.

PUSS'n BOOTS is Good Nutrition

America's largest selling cat food . . . adds the plus in health, beauty, vigor

Coast Fisheries, Division of The Quaker Oats Company, Wilmington, Calif.

(CLASSIFIED ADS—*continued from p. 44*)

Veterinarian wanted to assist in small animal practice; position leading to partnership. Virginia license required. Address "Box V 16," c/o JOURNAL of the AVMA.

Wanted—Positions

British graduate of University of Bristol, Jan. 1955, requires assistantship in mixed practice in New York State. Interested in operative surgery. Address "Box V 13," c/o JOURNAL of the AVMA.

(*Continued on p. 48*)

self feeding SODIDE

removes roundworms

Mixes palatably with dry feed.

•

With SODIDE pigs naturally worm themselves. When mixed 2 lbs. Sodide with 100 lbs. dry feed, Sodide can be effectively and easily administered in self-feeder. Feed exclusively for 24 hours, allowing no slop feeds during this time. You save by buying Sodide in the economical 25 lb. pail.

•

An E-plus Product. Easy to administer, proved effective, and economical. Blended of 50% Fluoride and 25% yeast in a palatable base.

•

Your Choice of Quantity
1 lb.
12-1 lb.
1-25 lb. pail

NLC

SERVING GRADUATE VETERINARIANS EXCLUSIVELY

THE
NATIONAL LABORATORIES
CORPORATION
KANSAS CITY

(CLASSIFIED ADS—*continued from p. 46*)

Veterinarian desires association with practitioner in small animal or mixed practice; licensed in Maryland, New Jersey, Pennsylvania. Some capital, if suitable proposition presented. Address "Box V 1," c/o JOURNAL of the AVMA.

Licensed in Illinois, Vienna graduate desires position or partnership. Ten years of broad experience with large and small animals; tops in sterility and some fields of surgery. Four years of experience in the United States; good English. Address "Box V 3," c/o JOURNAL of the AVMA.

Doctor of Veterinary Medicine, graduated from the Veterinary School, Lyons, France (graduates of this school are recommended by AVMA for recognition by State Boards and other agencies), Certificate de Bactendogie of the Pasteur Institute, Paris, desires position in research or industry. Special interests in virus research. Age 28, single, German; speaks German, English, French fluently. Address Richard Nanes, 59 High St., Nutley, N. J.

(*Continued on p. 54*)

D·L·V*

Hog Cholera Vaccine

- Confers Solid Long Lasting Immunity.
- Standard Dosage (2cc. of D·L·V - 10cc. Serum)
- Used with Maximum Results on Millions of Swine
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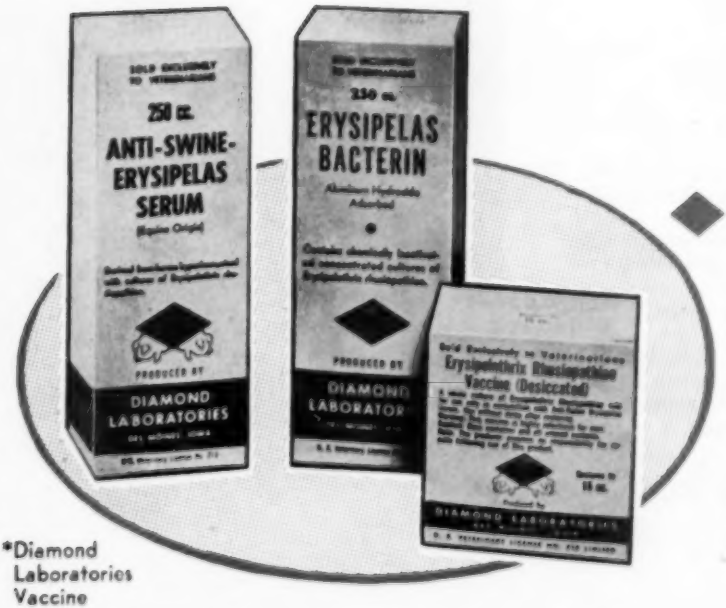

*Diamond Laboratories Vaccine

DIAMOND

Diamond Laboratories is exclusively owned and operated by Practicing Veterinarians.

Whether it's D·L·V, or one of the many other Diamond Products, you can be assured of quality products of the highest standard.

Quality Products of Dependability

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DES MOINES, IOWA

POLYOTIC*

Lederle Professional Line

POLYOTIC* INTRAMUSCULAR:
100 mg.-500 mg.-1.0 Gm.-5.0 Gm.

POLYOTIC INTRAVENOUS:
100 mg.-2.5 Gm.

POLYOTIC OBLETS®: 4's-6 x 4's

POLYOTIC CAPSULES: 50 mg., 25's-100's;
100 mg., 100's; 250 mg., 16's-100's

POLYOTIC TABLETS: 50 mg., 25's-100's;
100 mg., 25's-100's; 250 mg., 16's-100's

POLYOTIC MASTITIS OINTMENT: ¼ oz.

POLYOTIC COMPOUND MASTITIS OINTMENT: ¼ oz.

POLYOTIC OPHTHALMIC OINTMENT 1%:
6 x ⅛ oz.

POLYOTIC TOPICAL OINTMENT 3%: 1 oz.

POLYOTIC SOLUBLE (Tinted) POWDER:
¼ lb.-½ lb.-1 lb.-5 lb.

AVIANIZED® RABIES VACCINE (Canine):
1 dose-5 x 1 dose-10 doses

AVIANIZED RABIES VACCINE (Cattle): 10 doses

AVIANIZED CANINE DISTEMPER VACCINE:
1 dose-10 x 1 dose

ANTI-CANINE DISTEMPER SERUM AND ANTI-INFECTIOUS CANINE HEPATITIS SERUM: 20 cc. 100 cc.

INFECTIOUS CANINE HEPATITIS VACCINE: 2 cc.-10 cc.

BRUCELLA ABORTUS VACCINE: 1 dose-5 x 1 dose-
5 doses (25 cc.)

FELINE DISTEMPER VACCINE: 1 immunization (2 vials Vaccine, 2 vials Sterile Diluent, 2 cc.)

ANTI-FELINE DISTEMPER SERUM: 50 cc.

CARICIDE® Diethylcarbamazine TABLETS:
400 mg., 25's

DIETHYLSTILBESTROL SOLUTION: 10 cc.-50 cc.

LEPTOSPIRA CANICOLA-ICTEROHEMORRHAGIAE BACTERIN
Whole Culture Inactivated Vacuum-Dried.

Other products to be added.

*Trade-Mark

Lederle

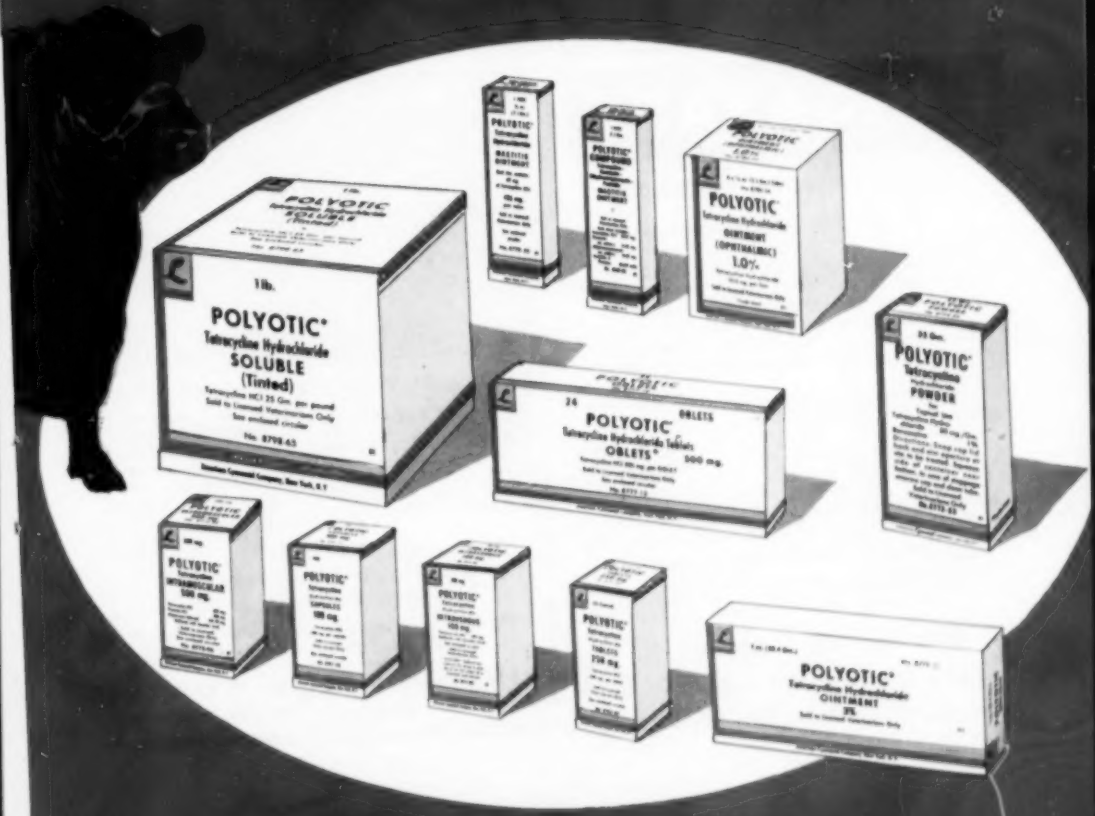
LEDERLE LABORATORIES DIVISION

Pearl River AMERICAN Cyanamid COMPANY New York

Tetracycline

Here is the newest of the broad-spectrum antibiotics, clinically proved and ready for intravenous, intramuscular, intrauterine, intravaginal, intramammary, conjunctival, aural and topical use.
No other antibiotic is available to the profession only in such a wide variety of dosage forms. No matter what route of administration, there is a POLYOTIC designed for proper professional application and for dispensing. Available to Licensed Veterinarians Only.

*Trade-Mark



Dr. Bunn — Veterinary Director of Merck-Sharp & Dohme International

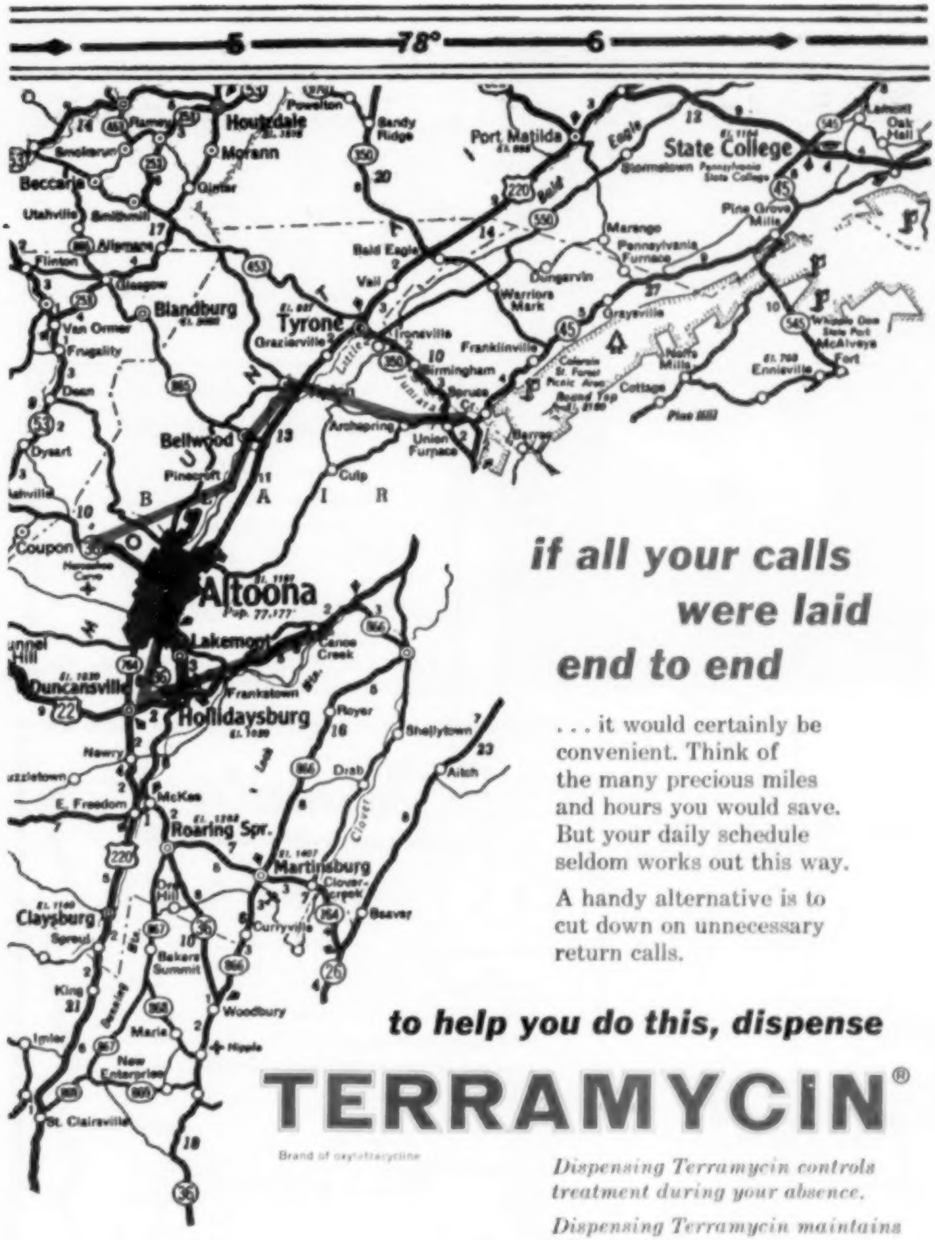
Dr. Carl E. Bunn (COL '51) has accepted the position of veterinary director of the Merck-Sharp & Dohme International division in New York. Dr. Bunn was formerly staff veterinarian in the medical division of Sharp & Dohme in West Point, Pa., under Dr. Samuel Scheidy, beginning in January, 1952.

Most birds move their wings in unison but the swift, a champion speedster, beats its wings alternately.—*Sci. News Letter, May 21, 1955.*

Veterinarian with 4 years of mixed practice experience interested in position leading to lease, partnership, or purchase of small animal practice in Baltimore area. Married, age 34; have Maryland license. Address "Box V 18," c/o JOURNAL of the AVMA.

Situation wanted leading to lease, partnership or purchase of predominantly small animal practice by experienced veterinarian licensed in New Jersey, Connecticut; age 31, married. Address "Box V 25," c/o JOURNAL of the AVMA.

(Continued on p. 56)



(CLASSIFIED ADS—*continued from p. 54*)

Experienced veterinarian, married, desires assistantship in small animal or general practice. Graduated from Hannover Veterinary College, 1944. Address "Box V 22," c/o JOURNAL of the AVMA.

Capable veterinarian with 10 years' practice experience seeks position with a future in mixed or small animal practice. Licensed in California; references. Initial salary secondary to opportunity for partnership or purchase in good West Coast community. Available at once. Address "Box T 25," c/o JOURNAL of the AVMA.

(*Continued on p. 58*)

Reproduction tests give added proof of Friskies' complete nutrition!

As professional kennelmen know, breeding presents diet problems that surpass those problems found in any other type of feeding. A dog food has to be right to satisfy the breeder – and to produce results such as have been attained at the Friskies Research Kennels on the famous Carnation Farms near Seattle, Washington.

In countless reproduction tests, proof of the value of a Friskies diet is evidenced in successive generations of champions, all maintained on Friskies.

Every food element dogs are known to need has been combined in the famous Friskies formula. Friskies Meal provides a moist feeding . . . Friskies Cubes, a dry feeding of *compressed* Friskies Meal, equally nutritious in every respect. For variety, feed both Meal and Cubes.

Friskies

Sold in 2, 5, 10, 25 and 50-lb. sizes.

ALBERS MILLING CO., DIV. OF **CARNATION COMPANY,** LOS ANGELES 36, CALIFORNIA

(CLASSIFIED ADS—*continued from p. 56*)

Wanted—Practices

Experienced small animal practitioner, age 30, wants to buy small animal hospital, or join partnership. Will locate anywhere in Southeast. Address "Box V 11," c/o JOURNAL of the AVMA.

Want to buy mixed practice in Connecticut or New York. Have substantial cash down payment. Must be available immediately. Address "Box V 20," c/o JOURNAL of the AVMA.

Wanted—a good small animal hospital doing at least $20,000 net annually. Address "Box T 13," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

Modern, established small animal practice for sale in eastern Pennsylvania; consists of waiting room, examination room, x-ray room, drug room, isolation ward, kennel room, and outside masonry runs. Large animal work available, if desired. Reason for selling, personal. Financing can be arranged. Address "Box T 10," c/o JOURNAL of the AVMA.

Small animal hospital for lease in northern California; modern, well equipped. Grossed $24,000 in last 12 months. Excellent opportunity with option to purchase. Address "Box V 21," c/o JOURNAL of the AVMA.

(*Continued on p. 60*)

(CLASSIFIED ADS—*continued from p. 581*)

Established mixed practice, 85% large animals, for sale in western Nebraska for price of new ranch-style building, combination home and hospital. Excellent location on busy U.S. highway one mile from business center of city of 4,000. Address "Box V 7," c/o JOURNAL of the AVMA.

Mixed practice for sale; clinic with x-ray, 10 outside runs, mobile chute, nice home on 28-acre farm just outside Alabama resort town of 4,000. Address "Box V 19," c/o JOURNAL of the AVMA.

Small animal hospital for sale; 80% small animals, 20% large. Six-room modern home attached. Located in Iowa town of 30,000. Price, $16,000. Address "Box V 23," c/o JOURNAL of the AVMA.

(*Continued on p. 62*)

An important new idea in Distemper-Hepatitis Immunization

distemper hepatitis

sin-jex

vaccine

Sin-jex **is the first Distemper-Hepatitis vaccine to utilize a vacuum dried *modified live virus* distemper fraction, with killed virus hepatitis fraction as diluent. *Sin-jex* gives more positive immunity against distemper: because the modified live virus distemper fraction elicits a more marked immunity response. *Sin-jex* is instantly reconstituted because of the R_L "reconsti-quick" feature. *Sin-jex* is a fine homogenous suspension which passes easily through a 22 guage needle. *Sin-jex* is easily administered with a single subcutaneous injection. *Sin-jex* may be given simultaneously with or without Anti-Canine Distemper-Infectious Hepatitis Anti-Serum. *Sin-jex* produces immunity in less than two weeks. *Sin-jex* may be used before or after weaning.**

Research Laboratories, Inc.
St. Joseph, Missouri

Available from

INDEPENDENT ETHICAL DISTRIBUTORS
selling to Licensed Veterinarians only
* TRADE MARK

(CLASSIFIED ADS—*continued from p. 60*)

Mixed practice for sale in Middlewest city of over 15,000. Grossing $18,000-$20,000 annually; 90% large animals; testing available. Includes modern downtown office, complete drugs, equipment, x-ray, etc. Address "Box V 14," c/o JOURNAL of the AVMA.

Outstanding small animal hospital for sale in wealthy southern California area; well located, modern, fully equipped, excellent buy. Requires $15,000 down. Other interest forces immediate sale. Address "Box V 17," c/o JOURNAL of the AVMA.

Small animal practice for sale in choice location, Santa Monica, Calif. No property involved, good lease, hospital, kennel equipment, and apartment furniture, $6,000 complete. Address "Box V 24," c/o JOURNAL of the AVMA.

Miscellaneous

Clipper Blade Sharpening—guaranteed to please you; factory trained. Our customers recommend us; 1,000 satisfied veterinarians. Avoid C.O.D., send money with blades, 75c per set; 24-hour service. Service Grinding Co., 903 Chicago St., Racine, Wis.

Pregnancy Diagnosis—in mare from 45th to 150th day. Write for vials and mailing tubes. Price: $7.00; 2 or more tests, $6.00 each. Pregnancy Diagnostic Laboratories, H. S. Lames, D.V.M., Dysart, Iowa.

Breeders' Sleeve—the disposable obstetrical sleeve. Package of 20 with detachable chest band, $5.00; lower wholesale prices. Free sample upon request. Breeders Equipment Co., Flourtown, Pa.

NOW! A NEW CONCEPT

IN CANNED DOG FOOD

FROM Gaines

Gaines' new Bodyguard Formula is the first to take advantage of the ability of Methionine to improve the Biological Value of dietary protein.

A Product of General Foods

Gaines' new **Bodyguard** Formula is the first to combine rich meat nutrients and other ingredients vital for balanced nutrition with *dl*-Methionine. It has been shown that added *dl*-Methionine can increase the Biological Value of dietary protein to an extent above and beyond that expected from its quantitative contribution to the essential amino acid level.*

The **Bodyguard** Formula's precise combination of vitamins, minerals, proteins and *dl*-Methionine gives dogs better food for growth, health, happiness. Its nourishment cannot be duplicated with other brands of dog food or choice cuts of meat.

You can recommend this new canned Gaines to dog owners with complete confidence in its nourishment, especially when young, aged, or recuperative dogs need high-quality protein in their diet.

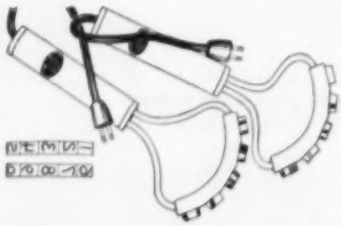
* The exact mechanism is not known, but *dl*-Methionine is ordinarily one of the last amino acids to be released from the protein and transmitted to the bloodstream in the course of normal digestion. It may, then, often be the limiting factor in the anabolism of protein. It is presumed that added *dl*-Methionine is absorbed more rapidly so that it reaches the cells at the same time as the other essential elements in protein metabolism. In this way more usable protein is made available.

Only Gaines has the

BODYGUARD

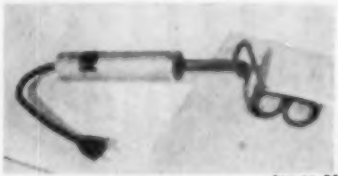
FORMULA

Gaines Canned Dog Food



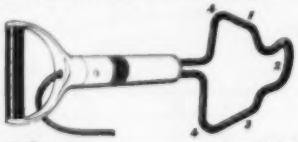
The letters are ⅞ inch, the most popular size. List Price of set, $69.50.

The New L & H All Purpose Electric Brander will brand any object that burns.
The price on Brander is $35.00. Letters or figures are $1.00 each, list.



$20.50 NET

It's Safe . . . No flame eliminates fire hazard—Completely insulated.
It's Fast . . . Heats Red Hot in 90 seconds! Quicker Heat Recovery—No Time Lost.



Universal Brander ..... List $42.50
1) 3 in. Bar; 2) 2 in. half circle; 3) 4 in. Bar; 4) corners may be used as running iron.

Deborner, without handle 29.50
Deborner, with handle ..... List 32.50

It's Simple . . .
Plugs into any 110 volt outlet or Standby generator. The tubular Rod type element is non-corrosive and self cleaning.

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SALES TO VETERINARIANS ONLY
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Index to Advertisers in This Issue

for dispensing and use by veterinarians only

for prevention and treatment of disease . . .

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5 Gm. of oxytetracycline hydrochloride (Terramycin®) activity per lb.

Terramix*-10

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. . . the growth-promoting, nutrition-enhancing, disease-preventing and therapeutic activity of Terramycin® in a form easy to dispense and easy to administer . .

. . . these products blend readily into the livestock and poultry rations (grain, silage, or mash), and can conveniently be incorporated in premixes.

in the NEW 5-lb. re-usable dispensing canisters with removable labels

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Department of Veterinary Medicine
PFIZER LABORATORIES, Brooklyn 6, N. Y.
Division, Chas. Pfizer & Co., Inc.

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the odds are in your favor
when you use—

SULBEN

for treating Calf Scours,
Foot Rot, Pneumonia,
Metritis, Coccidiosis,
Shipping Fever

No chances are taken when you use SULBEN . . . the sulfabenzamide preparation with a wide spectrum of efficacy against bacterial infections in animals and poultry. SULBEN is especially indicated for combating enteric conditions and those due to streptococcic, staphylococcic and *Pasteurella* organisms. In many cases a single dose is sufficient but dosage can be repeated if necessary. SULBEN is available in liquid for parenteral and oral use and in tabole, tabsule, hextab and toytab form.

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FOR PREVENTION AND TREATMENT OF

Shipping Fever

It is recognized that Pasteurella organisms play an important part in the losses incident to shipment or exposure of cattle. The probability of a primary virus cannot be overlooked. For this reason:

Anti-Hemorrhagic Septicemia Serum

(Lockhart)

is produced from mature cattle, proved to be immune to the complexes of "shipping fever." This PLUS VALUE accounts for the superiority of results obtained. In those areas where Corynebacterium is an important complication

Anti-Corynebacterium Pasteurella Serum

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is recommended. Either serum packaged in 100 cc., or 250 cc., or 500 cc. size, single or multiple packaged.

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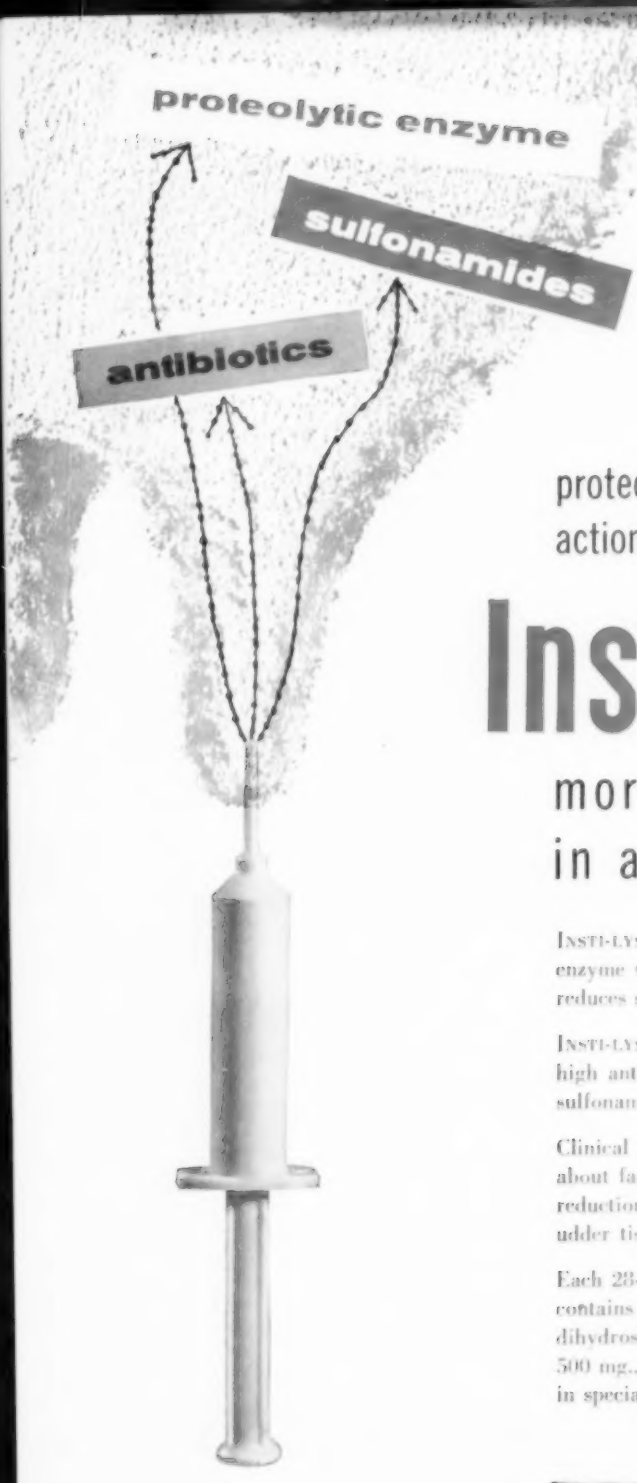
"Producers of Better Biologics for Graduate Veterinarians."



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action makes

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more effective in acute mastitis

INSTI-LYSIN contains papain, economical plant-origin enzyme which digests cellular debris and reduces swelling promptly.

INSTI-LYSIN, in special diffusible base, maintains high antibiotic udder levels. High sulfonamide content assures broad bacterial coverage.

Clinical tests have shown that INSTI-LYSIN brings about faster recovery, rapid and more complete reduction of swelling, with less damage to udder tissue than control therapy alone.

Each 28-cc. plastic syringe in dispensing box contains procaine penicillin G 500,000 units, dihydrostreptomycin 100 mg., phthalylsulfacetamide 500 mg., sulfathiazole 500 mg., and papain 50 mg. in special diffusible oil base.

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